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NON-OPERATIVE TREATMENT OF PERIPHERAL VASCULAR DISEASES*

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THE management of peripheral vascular diseases has been the subject of greatly increased interest during the past few years. This has been due, in part, to a realization that the vast majority of these cases, which formerly were problems of concern to physicians and surgeons only after some acute disturbance such as intermittent claudication or threatened gangrene had developed, are now appreciated to be preceded by a long period of a diminished circulatory reserve which should be detected and all efforts made to prevent the catastrophe of an amputation.

New procedures and new devices for estimating peripheral circulatory deficiency have been greatly improved, which, when properly applied, will materially aid in the detection of vascular disturbances long before the development of the clinical symptoms. We refer particularly to the use of the oscillometer and the thermocouple. The use of these instruments of precision, a pertinent history relevant to the aches and pains in the extremities and palpation of the pedal pulses; are slowly becoming a part of the routine of a complete physical examination.

There is today a greater desire as well as effort to try to prevent the amputation of extremities. This changing attitude of the medical profession has been brought about by many factors. The thousands of people unmindful of the fact that they have no pedal pulses have naturally brought up the question as to why the actual sufferers from peripheral vascular diseases cannot be tided over into a similar state of circulatory balance. Observations are accumulating which seem to justify the belief that the vast majority of our serious problems of peripheral vascular diseases result from a speeding up of the usual slow progressive occlusion of vessels or from an insult through trauma, infection or cold, which throw not only an acute strain on the collateral vessels but induce a period of vasospasm. This more or less sudden demand on the collaterals plus vasospasm of varying intensity give rise to the severe pain and gangrene unless the patient or the doctor or both can do something to tide them through these critical phases of the

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disease. A typical example is the cadaveric, vasospastic extremity resulting from a sudden arterial embolism or severe traumatism. The same process is repeated with varying clinical manifestations when the occlusion is only relatively rapid as a result of thrombosis of diseased arteries. A series of such events is witnessed repeatedly in cases of thrombo-angiitis obliterans. It is well illustrated by a patient who went through four distinct periods of threatened gangrene, each associated with a rapid occlusion of the tibial arteries, who then came to a circulatory balance adequate to give comfort the rest of his life, or in a patient with Raynaud's disease who, through extraordinary care, prevented a serious attack of angiospasm for a period of years. Observations such as these, together with the recent additions to therapy, such as sympathetic nervous system surgery and the use of passive vascular exercises, have made many surgeons change from the placid rôle of waiting for the time to amputate to an active rôle of trying to tide sufferers from peripheral vascular disease through to a stage of a comfortable circulatory balance such as we not infrequently see in people who have had a gradual yet extensive occlusion of their major arteries. We wish to consider these nonoperative forms of therapy which often help to prevent complications, relieve suffering, avoid amputation and establish a circulatory balance which will afford comfort.

From what has been said it is obvious that the great opportunity in this field rests in the early recognition of peripheral vascular deficiency. When it has become customary to study the peripheral vessels and to advise the patient when there is apparent a thin margin of safety in the blood supply of extremities, there are many things which can be done to avoid those critical periods of pain, infection, gangrene and amputation.

Many of these measures were popularized by Buerger and Allen, particularly the avoidance of injury and infection; the absolute necessity of keeping the feet warm; the immediate and most meticulous care of the slightest wound infection; keeping the skin of the feet in the most perfect condition by means of baths and oils; the avoidance of positions or active exercises which may tend to traumatize the main arterial pathways; a very large fluid intake; a judicial use of thyroid extract when the pulse is slow and the blood pressure low; the eradication of all foci of infection; abstinence from the use of tobacco; teaching patients the positions of optimum circulation when at rest, both lying and sitting down and Buerger or Allen active vascular exercises, *etc.* The difficulty in having these procedures carried out seems to be that few doctors have the time or patience to get patients to realize their importance and to establish them as a routine part of their lives. Many acute crises could be avoided if early diagnoses were made and these measures diligently practiced. The most meticulous observance of these measures of general care is among patients who have weathered periods of acute crises accompanied by severe pain or threatened gangrene. After such experiences it is not so difficult a matter to make patients realize the im-

portance of the many precautions and details of treatment that should be observed.

The time will certainly come when these measures will be employed extensively to lessen the first occurrence of the critical periods of peripheral vascular diseases. An advance will occur when doctors and patients evaluate the seriousness of the early disturbances of threatened occlusion. Then both time and effort will be devoted to tiding people, afflicted with peripheral vascular diseases, through to a state of more or less stationary circulatory balance consistent with activities which, of course, may have to be reduced. At the present time, only a few physicians are regarding these early disturbances seriously or doing anything particularly about them except when the critical periods of pain or threatened gangrene develop, and even then many patients are allowed to return to their former routine of life with only the most vague notions of what to do to prevent a recurrence of such experiences.

Preventive measures are rarely formulated until after a long period of actual combat with the fully developed disease. This has been true in most of our medical problems and seems to be necessary in order to kindle the interest from which eventually spring efforts in the field of preventive work. We are now at the stage of the beginning of active interest in combating the fully developed ravages of peripheral vascular diseases. Neither the public nor the medical profession is satisfied with amputation as the sole therapeutic effort. Waiting for the time to amputate and then deciding at what level to do it is not conducive to the development of preventive measures.

Much progress has been made in the treatment of the acute phases of the peripheral vascular diseases; notably the most meticulous care of wounds, infections and gangrenous areas; rest; proper level of the affected extremity; hypodermoclysis with large quantities of Ringer's solution daily; intraduodenal administration of large amounts of hypertonic salt solution; intravenous administration of sodium citrate solution; constrictive hyperemia; intravenous administration of sodium iodide or calcium chloride; the use of typhoid vaccine and other forms of foreign protein therapy; thermopyrexia; the use of vasodilating drugs; the ligation of the femoral vein; periarterial sympathectomy and sympathetic ganglionectomy; ligation or excision of the femoral artery and paralysis of the peripheral sensory nerves.

Comment on two of these procedures is pertinent. The importance of keeping a resting extremity in the position which will secure for it the maximum circulation cannot be overstressed. The elevation of extremities as an essential part of the therapy has undoubtedly been responsible for a large number of amputations. This practice is a glaring example of unbelievable blindness to an universal instinct of patients, who not only refuse to keep the extremity elevated but often alternately hang it over the side of the bed and put it back. We have previously stressed the great dangers of resting the extremity in an elevated position, these have been confirmed by calorimetric studies.

Air superheated beyond the level of the body temperature has recently been shown by Freeman to be actually harmful. We have been under the impression for some time that the electrically heated cradles increased the pain and the progression of gangrenous extremities. Freeman has shown that excessive temperature makes the tissues call on the circulation for greatly increased metabolic processes. It is obvious that the blood which reaches an affected extremity should be effective and oxygen carrying as long as possible. We are using flannel or large wool lined stockings heated to just above average skin temperature before being applied. Definite harm results from a fluctuating environmental temperature about affected extremities. A draft of cold air over a leg in a critical phase from arterial disease will cause immediate blanching of the skin. The doors and windows should be closed before exposing affected extremities for dressings, studies, or treatments. It is desirable to keep the environmental temperature as nearly that of the skin as possible during the acute phases of peripheral vascular diseases. Our problems in this field of work in the winter as compared with those in the summer time illustrate the importance of the effect of subnormal environmental temperatures. It therefore appears desirable to keep patients with threatened or actual gangrene in rooms in which the temperature approaches that of the skin. Certainly wide fluctuations of environmental temperature either above or below that of the normal body (surface) temperatures should be avoided. Of the other measures mentioned many need no discussion. Some are of obvious value; the efficacy of others must be determined. The importance of some depends upon more or less specific problems with which one is confronted.

While the use of extreme degrees of heat, as caused by the electrically heated cradles, has been disapproved of in this paper it should be stated that the use of cold air to produce the passive vascular exercises may be harmful because of the vasospasm which it produces. This difficulty has been obviated by the use of air preheated to a level slightly above body temperature (102–105 degrees Fahrenheit).

A working classification is desirable but one should not classify his cases too rigidly as any long-standing case will usually exhibit some of the characteristics of two or more of the various types of peripheral vascular diseases. What was in the beginning a true case of Raynaud's disease may show later organic obliteration of many peripheral vessels due to thrombosis and fibrosis. An arteriosclerotic case may develop a sudden occlusion of various vessels due to thrombosis or arterial embolism. Any case of primary organic disease which develops a sudden critical period such as may be brought on by thrombosis, embolism, rupture of a vessel, injury, exposure to cold, *etc.*, always exhibits an element of vasospasm which may be temporary but still a very vital factor during the period when gangrene is so likely to develop (Table I).

TABLE I

CLASSIFICATION OF PERIPHERAL VASCULAR DISEASES

(A) *Primary Vasomotor Disturbances*

a. Vasoconstrictor disturbances

(1) Raynaud's disease
(2) Acrocyanosis (acro-asphyxia chronica; acroparesthesia; sclerodactylia)

b. Vasodilatory disturbances
(1) Erythromelalgia
(2) Acute painful osteoporosis (?)

(B) *Primary Organic Diseases of the Arteries**

a. Traumatic (chemical and thermal)
(1) Embolism and simple thrombosis
(2) Arteriovenous aneurysm
(3) Phenol and all caustics
(4) Frost bite

b. Inflammatory (toxic)
(1) Thrombo-angiitis obliterans
(2) Specific arteritis (syphilis, tuberculosis, peri-arteritis nodosa pyogenic)
(3) Nonspecific arteritis (exanthemata, typhus, typhoid, pneumonia)
(4) Nonspecific arteritis (chronic toxemia, ergotism)
(5) Endarteritis obliterans (cause undetermined)

c. Degenerative changes
(1) Arteriosclerosis (senile, diabetic and Mönckeberg)

We have made extensive use of intermittent *negative* and *positive* environmental pressures (Passive Vascular Exercises) in the treatment of the acute pain and threatened or actual gangrene due to peripheral vascular diseases. These exercises have been used in conjunction with other general measures which are appended.

GENERAL DIRECTIONS FOR HOME CARE OF THE FEET

Ambulatory Treatment

1. Wash feet each night with neutral (face) soap and warm water.
2. Dry feet with a clean soft rag without rubbing the skin.
3. Apply rubbing alcohol (70 per cent) and allow the feet to dry thoroughly, then apply a liberal amount of vaseline or toilet lanolin and gently massage the skin of the feet.
4. Always keep your feet warm. Use woolen socks or wool lined shoes in the winter and white cotton socks in warm weather. Use a clean pair of socks each day.
5. Use loose fitting bed socks instead of hot water bottles, electric heaters or any other form of mechanical heating devices.
6. Wear properly fitting shoes and be particularly careful that they are not too tight. Use shoes made of soft leather and without box toes.
7. Cut your toe-nails only in very good light and only after your feet have been cleansed throughly. Cut the toe-nails straight across.
8. Do not cut your corns or calluses.
9. Do not wear circular garters.
10. Do not sit with your legs crossed.
11. Do not use strong antiseptic drugs on your feet. Particularly never use tincture of iodine, lysol, cresol or carbolic acid.
12. Go to your doctor at the first signs of a blister, infection of the toes, ingrowing toe-nail or trouble with bunions, corns or calluses.
13. Drink at least four quarts of water each day.
14. Eat plenty of green vegetables and fruit in an otherwise well-balanced, liberal diet, unless you have been ordered to follow some special regimen.

* Vasospasm may occasionally play a constant part in the picture of the obliterative diseases; it always plays a temporary but very important part during the sudden critical periods of all vascular diseases.

15. Do not use tobacco in any form.
16. Have some member of your family examine your feet at least once each week.
17. Carry out your exercises prescribed by your doctor exactly as you were taught to do them in the clinic. Do them regularly and faithfully.
18. Come to the clinic for Passive Vascular Exercise treatments as directed.

HOSPITAL ROUTINE FOR PATIENTS WITH PERIPHERAL VASCULAR DISTURBANCES

1. Bed should be fitted with a Balkan frame with hand grips to aid patients in carrying out exercises and permit patients to move around in bed easily.
2. Affected extremity or extremities should be placed on a pillow in the position which affords optimum circulation to the part. Heels should extend over the edge of the pillow to prevent excessive pressure from the mattress.
   Position of optimum circulation as determined in the Vascular Clinic,

(A) Right foot,

   (*a*) Recumbent position.....
   (*b*) Sitting position.....

(B) Left foot,

   (*a*) Recumbent position.....
   (*b*) Sitting position.....

3. Large, doubly insulated cradle over the legs and feet, when necessary to relieve pressure of bed covers. Cradle should *not* be fitted with electric bulbs or other mechanical heating device, but the legs kept warm with large woolen or flannel stockings, or by keeping the room warm.
4. Well-balanced, liberal diet.
5. Drink at least four quarts of water each day.
6. Patients should abstain from the use of tobacco in any form.
7. Daily cleansing of the feet. Careful application of neutral (face) soap and warm water followed by alcohol (70 per cent). Dry feet without friction. Apply vaseline or bland oils to roughened or irritated areas. Special care should be given to the toe-nails and thick calluses.
8. Ulcerated areas on the extremities should be specially treated.
9. Use only non-habit forming drugs for the relief of pain in the extremities.
10. Postural treatments as advised by Buerger and modified by Allen should be carried out as directed.
11. Passive Vascular Exercise treatments as directed.
12. All diabetic patients are simultaneously under the care of the Medical and the Surgical Services.
13. The temperature of the patient's room should be kept at 80° F. or slightly higher and no fluctuations of this temperature should be permitted during examinations, treatments or dressings.
14. All important clinical studies on the patients with peripheral vascular disturbances should be made in the Vascular Clinic of the Dispensary.

The effect of decreased atmospheric pressure upon the peripheral circulation has long been known. The fundamental idea of applying alternately *negative* and *positive* pressure to an extremity to increase the arterial circulation was expounded and put into actual clinical practice more than 50 years ago by Edgar Bluck, of Hamstead, England. In July, 1932, Dr. Louis G. Herrmann designed and built the original "Pavaex" units used to establish this method in our clinic and soon presented evidence that it was a valuable adjunct to conservative methods which we have advocated for many years.

The mechanical part of the apparatus has been modified and improved and studies made of the effect of various alterations of the curve of pressure changes and its actual influence upon the circulation of the extremity.

We found that approximately 80 Mm. *negative* and about 20 Mm. *positive* pressure with an alternation rate of two to four cycles each minute brought about the greatest increase in the arterial circulation and caused the least damage to the intimal lining of the arteries, in patients with organic changes in the peripheral arteries. Elevation of the foot several inches above the level of the heart was found to be more satisfactory than the use of larger amounts of *positive* pressure to free the extremity of venous blood. Certain variations of these pressures have frequently been necessary as, for example, when treating the cyanotic or static type of peripheral vascular disturbance which is associated with extensive arteriosclerosis, it has been found advisable to decrease the amount of *negative* pressure to about 60 Mm. and to increase the *positive* pressure to about 40 Mm.

The frequency or length of the "Pavaex" treatments must depend entirely upon the urgency of the condition which is being treated. Acute arterial occlusion either by operation, thrombosis, trauma or embolism must be considered as a surgical emergency and the "Pavaex" treatments must be given as often as necessary to keep the extremity alive until an adequate collateral circulation has become established. The number of hours of actual "Pavaex" treatment varies from four to eight hours each day, while the less urgent and ambulatory patients receive from seven to 15 hours of "Pavaex" treatment each week.

We would like to recite some of our observations with "Pavaex" therapy and present our ideas concerning the usefulness of this therapeutic adjunct to the treatment of various types of peripheral vascular disorders. The most startling results have been obtained among those patients who were suffering from a more or less sudden occlusion of the major arterial pathways to an extremity. There are also the patients in which temporary vasospasm of varying degrees plays such an important rôle in the ultimate outcome of the conditions; well illustrated by the ivory-like skin in certain cases of embolism, sudden thrombosis, ligation or rupture of a main artery in relatively young people when we know the collateral vessels are adequate if only they could be made to function. Further definite proof can be found in the reports of "Stupeur Arterielle" during the World War, the work of Leriche and the more recent report by Oughterson of the relief of this sudden vasospasm by immediate lumbar sympathetic ganglionectomy.

Our experiences in treating these acute crises of peripheral vascular accidents have been for us the most thrilling observations in our clinic for many years. In every instance of acute major arterial obliteration in which there has not occurred obvious death of tissues we have been able to relieve the spasm and draw blood into the extremity in an astonishingly short time with quick relief of pain and the prevention of gangrene. This has been accomplished even when sensation in the extremity has been absent or markedly diminished. When "Pavaex" treatments were started a striking case was that

of a woman 37 years of age with transient auricular fibrillation associated with mitral stenosis. Treatments were begun five hours after an embolus had lodged at the bifurcation of the right common iliac artery. The pain in her leg, which was agonizing and not relieved by large doses of morphia, was completely relieved within 15 minutes by passive vascular exercises and the color of the leg was changed from an ivory white to that of an active hyperemia. After a few days another embolus lodged in the left popliteal artery and the subsequent circulatory deficiency in the leg was relieved in less than five minutes. During the next two weeks the "Pavaex" treatments which at first were given at intervals of 60 minutes for periods of 30 minutes were gradually diminished in frequency until only five treatments of one hour were given each day. Three weeks after the first accident all treatments were discontinued and she has continued to be well since February, 1934. Pedal pulses have returned in the left foot but there is no palpable pulse in the entire right thigh or leg. The arterial circulation is adequate in both lower extremities. Operative procedures of either embolectomy or lumbar ganglionectomy have been avoided. The dangers of subsequent thrombosis and continued neurocirculatory imbalance would certainly have continued after embolectomy. This case is typical of many other experiences we have had with cases of arterial embolism, peripheral aneurysms and sudden traumatic interruption of the major arteries.

The value of "Pavaex" therapy diminishes as we approach the other extreme of peripheral vascular disturbances where occlusion of the arterioles occurs unassociated with sudden increases in the pathologic process and consequently uncomplicated by vasospasm or neurocirculatory imbalances. In some conditions there seems to be a condition of vasoparalysis as contrasted with vasospasm. In such cases the foot and toes usually remain congested, red or cyanotic in nearly all positions with respect to the level of the heart and usually there is great pain. The picture is frequently complicated by serious disturbances in the veins. The primary and secondary arteries may or may not be occluded. Often the posterior tibial pulse is palpable and oscillometric readings may show evidence of some arterial circulation. In such cases the value of "Pavaex" treatments is not established and may rank as secondary in importance to the general procedures which have been outlined, if, indeed, not actually harmful.

There are all stages of progression of peripheral vascular diseases. Whenever the occlusive or thrombotic arterial processes outbalance the ability of the collateral vessels to furnish adequate arterial blood and also upset the normal, delicately adjusted vasomotor mechanism, then we have minor or major crises in which "Pavaex" therapy forms a most valuable adjunct. To be more specific, such conditions usually are present in cases of intermittent claudication together with other types of pain associated with rapid blanching of an elevated extremity. The main factor which frequently disturbs the circulatory balance after slow arterial occlusion is cold, which disturbs the normal vasomotor balance and prevents an adequate adjustment of the circulation.

The use of "Pavaex" therapy in the presence of open ulcers, acute infection or gangrene, calls for definite judgment. An old principle usually attributed to Larry specifies rest and nontraumatization as being very beneficial in the healing of wounds and in combating infections. It is obviously inadvisable to apply the massage by strong *negative* and *positive* pressures to acutely inflamed tissues. It is impossible to make a definite statement as to when it is safe to use it in connection with these various complications of peripheral vascular diseases. Common sense judgment must, to a large extent, be relied upon. In the presence of dry gangrene without obvious infection and without active migratory phlebitis we do not hesitate to employ it nor do we hesitate to use it in the presence of open superficial wounds provided the pressures are so regulated that they do not cause bleeding of the granulating tissue. With these precautions we have not yet encountered any trouble from the lighting up or the spreading of infective processes in a very large series of patients who have received active treatment.

True thrombo-angiitis obliterans is a condition diagnosed about ten times as often as it actually occurs. Because of the obvious inflammatory nature of the disease, involving not only the arteries but the veins and nerves, we have had our difficulties both with regard to selection of pressures as well as the length of time of application of the "Pavaex" therapy. When there is acute superficial migratory phlebitis or intense rest pain out of proportion to what one can see, the inflammatory process appears to be so active that "Pavaex" therapy may do harm. The spreading inflammation and the vasomotor imbalance are indicated by the intense pain and the remarkably rapid play of skin colors when the foot is subjected to changes of temperature or to alterations of position with respect to the level of the heart. In the quiescent phases of this disease when the intense rest pain is gone and the thrombo-inflammatory processes apparently inactive, the addition of "Pavaex" therapy has been beneficial. When the sufferers from this disease are fortunate enough to reach the stage of complete healing of the thrombo-inflammatory processes, nearly always accompanied by complete obliteration of the major arterial vessels, they are usually on pretty safe ground and get along comfortably with the continued observance of the great care learned by experience during their long siege of suffering. However, these so called healed or stationary cases may be handicapped in their work and exercise from intermittent claudication and foot pains. In such cases the use of "Pavaex" therapy has been very beneficial but even then we have employed caution in the degrees of pressures used, lest the inflammatory processes be lighted up.

The great problem with patients in the acute or threatening stages of thrombo-angiitis obliterans is to get them to realize the seriousness of the disease and to adjust their lives to the loss of one or more years devoted entirely to the business of trying to get the inflammatory processes healed and a comfortable circulatory balance established. This attitude toward the problem would greatly enhance our results and would undoubtedly lessen the

so frequent extension of the thrombo-angiitic process to cardiac and other visceral vessels.

In patients with peripheral arteriosclerosis associated with diabetic mellitus in which we so frequently see ulcerations, infections and gangrenous toes, the arteriolar bed is usually fairly good and "Pavaex" therapy has enabled us to establish an adequate circulatory balance. It, of course, must be used with great discretion and absolute willingness to vary the amounts of pressure as indicated by the condition of the foot. When there is dry gangrene, with no active inflammation, pressures of 80 Mm. *negative* and 20 Mm. *positive* are generally used. If there is a little more active inflammation we may begin with a *negative* pressure of 60 Mm. with no *positive* pressure; depending on elevation of the extremity, during treatments, for aid in the return of the venous blood. When there is definite venous engorgement, without acute spreading inflammation, we sometimes use *positive* pressures of as much as 30 to 40 Mm. To state the problem as a general principle—nobody will deny the benefit derived from active hyperemia in combating inflammatory processes, nor the harm of active massage in such processes. Procedures of therapy often put these two principles into antagonistic action and one has to decide at what point one must stop in order to secure the maximum benefit to the patient. An analogy of these antagonistic forces is seen in the treatment of an infected hand, in that one does not usually continue absolute rest throughout treatment at the expense of a functionless hand in order to avoid the danger of spreading the infection from manipulating the fingers. Active hyperemia of passive vascular exercises has been used in a large series of such cases and we feel it has been of immeasurable aid in combating all forms of gangrene, as well as ulcerations, or low-grade infections. Among these disturbances we have had many of our most striking results, associated with a quick healing of wounds and a rapid demarcation of the gangrene.

With a prolonged use of over 100 Mm. *negative* pressure, we have produced petechiae in the skin and have observed actual rupture of intimal surfaces of the smaller blood vessels. The average *negative* pressure should be approximately 80 Mm. There are a few theoretical dangers associated with the use of Passive Vascular Exercise therapy but with reasonable judgment it has proved to be a very safe and efficient addition to the therapy of peripheral vascular diseases, in an experience of more than 16,000 hours of treatment, to over 300 patients with obliterative arterial diseases of the extremities.

ARTERIAL SPASM IN THE EXTREMITIES*

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The element of spasm in peripheral arterial disease has long been known, but its importance only recently recognized. All of the most careful students of the circulation have realized that a purely mechanical explanation of it was inadequate. Thus Harvey, in his dissertation "De Motu Cordis et Sanguinis," definitely links the force of the heart beat with the ability of the peripheral vessels to change their caliber as the two factors controlling the circulation. In another place he expresses this idea in a very specific form[1] as follows: "It is manifested that the blood in its course does not pass with the same celerity, neither with the same force in the same places and at all times. . . . In fear and under a sense of infamy and shame the face is pale, but the ears burn as if for the evil they heard or were to hear." To John Hunter[2] we are indebted for the first proof of contractility in the arteries. He not only demonstrated this property, but, by measuring the relative proportions of contractility and elasticity in the various arteries, he established the significant fact that the small arteries chiefly possess this ability of regulating by their contraction the amount of blood going to the regions they supply, while changes in the caliber in the larger arteries are principally due to elasticity. We can but marvel at the accuracy of these deductions based upon experiments aided only by the simplest of instruments and carried out before the time of the Revolutionary War, fifty years before the histologic proof by Henle that smooth muscle fibers were present in the arterial walls. The presence of nerve filaments extending to the arteries was an anatomic fact of indefinite antiquity even in the eighteenth century, and their action in constricting the arteries was considered a necessary hypothesis. Only its actual demonstration was lacking, as is well illustrated by Haller's[3] statement in summarizing his extensive experiments. "The sudden changes produced in the circulation by the mind, the erection of the penis, of the clitoris, of the nipples of the breast; the changes of color in the cheeks; cold and heat, tremors, inflammations, obstructions, the strong motions which violent pains or wound of the nerves often occasion. All these facts considered render very probable the action of the nerves on the arteries; since it, by this, appears that they have certainly a great power, either of retarding or accelerating the circulation. But I have never been able by any experiment to find, by observation, that irritating the nerves created any change in the velocity of the humours contained in the vessels of the circulation. I have indeed observed once or twice that by irritating the nerves it revived the action

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of the blood and restored the motion of it in arteries that were cut which had ceased to supply any. But I have had great reason to attribute this effect to some mechanical action." Such was the situation for another century until within the same decade epochal discoveries were made in the physiologic and in the pathologic fields. Claude Bernard[4] demonstrated the vasomotor control by sympathetic nerves over the peripheral circulation in 1851. In 1858, Brown-Sequard[5] made the following deduction: "In those cases of gangrene in which no obstruction has been found after death in the vessels of the dead parts, it is extremely probable that a long persistent spasm of the blood vessels has existed." Four years later Raynaud,[6] in his classic monograph on local asphyxia and symmetrical gangrene in the extremities, clearly showed that spasm could lead to gangrene. From this time we have known of arterial spasm as the cause of a relatively uncommon condition bearing Raynaud's name. But until recent years we have, aside from this, practically ignored it clinically. With the reawakening of our interest in the sympathetic nervous system since the war, methods for the clinical study of vasomotor phenomena have been developed. The data obtained have necessitated a complete revision of our conception of the peripheral circulation merely as a mechanical hydraulic system. In addition to its occurrence in Raynaud's disease, arterial spasm is often a critical factor in the common organic arterial diseases. It is a frequent accompaniment of nerve irritation whether in the peripheral nerves or in the central nervous system, and it is an important sequel of trauma.

SPASM IN ORGANIC VASCULAR DISEASE

When one of the main arteries in the extremity is occluded by organic disease, the effect on the local circulation varies enormously. It has long been common knowledge that you may have complete obliteration of pulsation in one or even both of the major arteries of the foot without impairing the viability of the part or even without causing any very noticeable symptoms. In such a case the condition of two classes of vessels becomes of paramount importance; namely, the collateral circulation and the smaller terminal arteries. In advanced noninflammatory occlusive disease such as that typically seen in arteriosclerosis occlusiva, either senile or diabetic, it can easily be demonstrated that associated with the circulatory deficiency there has been an inhibition of vasoconstrictor tonus. If the vasomotor nerves to the part are temporarily paralyzed by spinal, general or local anesthesia in such a case, there is no improvement in the circulation. Nature apparently has provided some method under these circumstances of overcoming even the physiologic degree of vasoconstriction.[7] On the other hand, in many instances of inflammatory or irritative lesions of the arteries, it is possible to demonstrate a striking improvement in the deficient circulation when the sympathetic innervation is temporarily or permanently abolished.[8] Arterial spasm in such instances often does play the determining rôle in making a reduced circulation insufficient to maintain the viability of the part.

We believe the explanation for this can be deduced from the nature of the pathologic lesion in the two instances. In arteriosclerosis occlusiva the process is primarily a degenerative one and is limited to the intima and media of the vessels. In thrombo-angiitis obliterans, on the other hand, there is an inflammatory reaction extending through the wall of the vessel. In fact, this is often so marked that the vein, artery and accompanying nerve trunks are glued together in one mass by it. The irritation of sensory sympathetic nerve fibers is probably the point of origin for the afferent impulses which cause the marked vasoconstrictor reflex. The motor impulses then cause a constriction in the diseased vessels from which the reflex arose, a result of no great importance, but they also cause a constriction of the anastomotic and terminal arteries on which the viability of the extremity distal to the arterial obstruction depends. This is the serious element about vasoconstrictor spasm associated with an organic disease of the arteries. It is probably true that recent embolism or rapid thrombosis usually evokes a similarly irritative vasoconstriction of the collateral circulation.

The recognition of this element of spasm in organic arterial disease is of great importance as its eradication enables us to improve the collateral circulation. The best method of accomplishing this is still somewhat in doubt. Peri-arterial sympathectomy does not permanently eradicate such spasm. Lumbar ganglionectomy does do so but is a major operative procedure. The injection of foreign protein and other fever-producing drugs will result in vasodilatation for a few days but are definitely contra-indicated in patients with any degree of arteriosclerosis, on account of the danger of thrombosis. We have been very hopeful that the method of passive vascular exercise as outlined by Doctor Reid would materially assist in overcoming such spasm just as the muscle spasm about an inflamed joint can be gradually overcome by extension. Possibly the addition of a vasodilating stimulation by warming the blood in the simple method devised by Landis and Gibbon[9] during the time the alternate suction and pressure is being applied to the involved extremity may further assist in this regard. In the common organic peripheral arterial diseases it is chiefly in thrombo-angiitis obliterans that continued spasm plays a particularly significant rôle and we wonder whether the added load put upon the vessel whose walls are involved in the inflammatory reaction would tend to light up the active phase of the disease. None of the vasodilating drugs that have yet been used has succeeded in producing a lasting obliteration of this vascular spasm. Up to the present we still feel that lumbar ganglionectomy in carefully selected cases offers these patients the most complete and lasting vasodilatation of the collateral circulation. In brief, then, the treatment of arterial spasm superimposed upon organic vascular disease still has to be individualized for each case.

RAYNAUD'S DISEASE

The classic example of arterial spasm is that seen in Raynaud's disease. The paroxysmal nature of the attacks in the absence of evidence of organic

arterial disease makes the diagnosis of the outspoken cases of this malady relatively simple if no cause for vasomotor irritation can be found. Usually attacks can be initiated by immersion of the hands in cold water. Women are predominately the sufferers of this disease. From the time of Raynaud's original description, the theory of increased vasomotor activity had not been seriously questioned in the etiology of this condition until recent studies of Lewis and his coworkers.[10] He advanced the theory of a local fault in the periphery independent of vasomotor innervation. Certain it is that psychic and reflex stimuli can induce true attacks of the disease. On the other hand, there are apparently typical cases in which the angiospasm is not fully released by temporary paralysis of the vasomotor nerves but is abolished by heat. Quite possibly these divergent views in regard to the underlying mechanism can be partially harmonized by the recent discovery of Smithwick, Freeman and White.[11] They found that small amounts of adrenalin produced an attack of angiospasm in the extremities of patients with Raynaud's disease even after complete sympathetic denervation. Possibly, then, the local fault of Lewis may be a hypersensitivity to circulating adrenalin in these patients. We have not seen any patient with what appeared to be uncomplicated Raynaud's disease whose angiospasm was not at least diminished by a temporary or permanent paralysis of the vasomotor innervation. In regard to treatment, most of the cases of Raynaud's disease that we see in our clinic are not sufficiently severe to warrant urging a major operation. They are inconvenienced by the attacks but when they protect themselves from the cold they can carry on a fairly normal life. If such a person can move to a warm climate, we advise her to do so. In addition to such protection from cold, we use Kerr's method of treatment by recourse to the reflex hyperemia which follows hypercooling the extremity.[12] When this is insufficient, or if the sensory acuity or the nutrition of the fingertips is threatened, then we advise dorsal ganglionectomy after proving that temporary denervation improves the circulation.

When one becomes interested in spastic arterial disease, he finds a great number of individuals who have minor degrees of angiospasm in the hands, almost exclusively in women. In these individuals, the degree of vasoconstriction in the upper extremity may be as marked or even more so than in the lower extremity. The milder examples of this type merely show such an accentuation of the vasomotor gradient in the hand (CHART 1). From this, however, there are all gradations up to true ischemic attacks in one or more fingers which have been described under the term "dead fingers." Just what the relationship of these more numerous lesser degrees of angiospasm is to true Raynaud's disease is uncertain. There is no evidence to suggest that these individuals are likely to develop a severe form of Raynaud's disease, but there is no line that can be drawn to separate their condition from the more pronounced syndrome.

ANGIOSPASM SECONDARY TO FUNCTIONAL AND ORGANIC NERVOUS DISORDERS

Anyone who has treated a considerable number of cases of old poliomyelitis will recall the usual coolness and blueness of the foot on the involved side at a moderately cool room temperature. This is a form of increased vasoconstrictor tonus as compared with the normal side and is not due to organic arterial disease as can easily be demonstrated by a temporary vasomotor paralysis. We have found that many organic diseases of the central nervous system associated with irritation or scar tissue formation may have an outstanding vasomotor component. We have seen this phenomenon also as a localized condition in functional nervous disorders. Irritation of the periph-

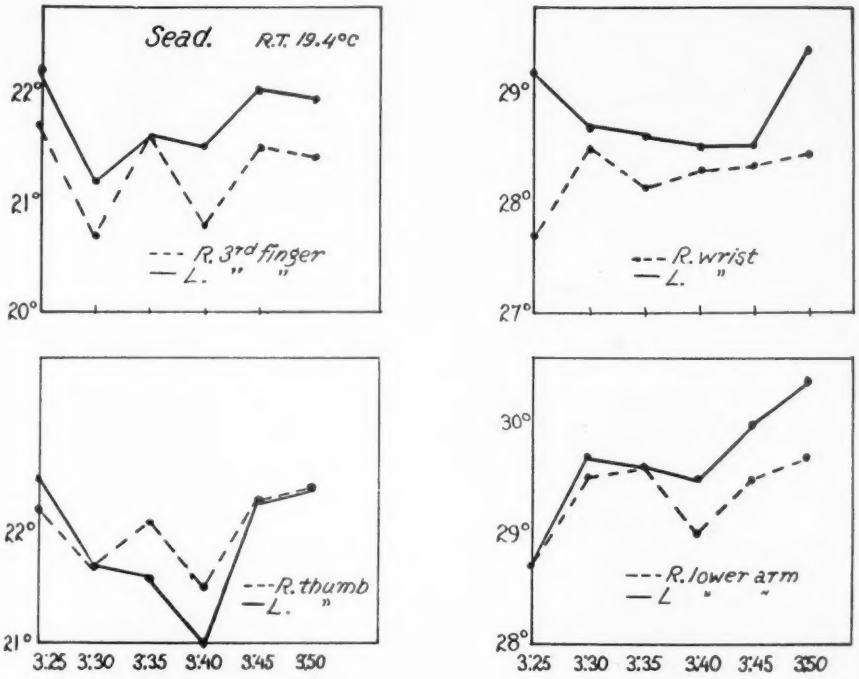

CHART 1.—Temperature readings of an exaggerated vasomotor gradient in the upper extremities in a patient without other evidence of vascular disease. Note the extremely low temperatures of the fingertips. This patient had always known that her hands were colder than those of most people. No evidence, however, of attacks resembling Raynaud's disease.

eral nerves or their roots such as is found with cervical rib, causalgia, or neuroma formation may be associated with marked angiospasm.

The following recent case is an excellent example of vasomotor neurosis of this type.

CASE REPORT

H. W., No. 94126, female, aged 45. Fifteen months ago this patient first noted vague numbness and tingling of the right hand which disappeared in a few days. She had a similar attack of short duration six months ago. Three weeks ago she began having intense numbness and tingling of the whole right hand extending up in an irregular fashion on to the wrist. Associated with this she had noted that the hand became bluish and at other times pale. With the attack she had pain in the hand. Recently she had noted

some pain in the right shoulder also. Her right hand had become extremely sensitive to cold, and heat relieved the numbness and pain. For the last ten days she had had to get up several times during each night on account of these symptoms, putting her hands into warm water, which promptly relieved the pain. Also at times during the day she had kept the hand wrapped in flannel.

Past History immaterial as far as the present illness is concerned.

Physical Examination.—Right hand paler and cooler than the left; radial and ulnar pulses good on both sides. Tenderness over the right seventh cervical transverse process. Hypoesthesia on the palmar surface of the right hand and fingers; hyperesthetic area in

FIG. 1.—H. W. Roentgenogram of cervical region shows a hypertrophic spine situated at the tip of the right seventh transverse process extending forward, its shadow extending under that of the first rib. The lower borders of both seventh cervical transverse processes have been outlined with pencil on the film.

the right thumb and wrist. Also area of hyperesthesia over the right shoulder and right antecubital fossa. Weakness on movements of the right hand and to a lesser extent of the right forearm. Immersing the hands in cold water at 15° C. shows marked sensitiveness of the right hand and causes some color changes. Recovery of the hands, however, from the cold bath is at approximately the same rate. Exposure of the right hand to cold water produces the same symptoms as the patient's attacks.

A radiograph showed a hypertrophic spine extending forward from the tip of the right transverse process. It is exactly at this point that the patient experienced pain (Fig. 1).

Preoperative Diagnosis.—Vasomotor neurosis due to pressure on the brachial plexus of an elongated right seventh transverse process.

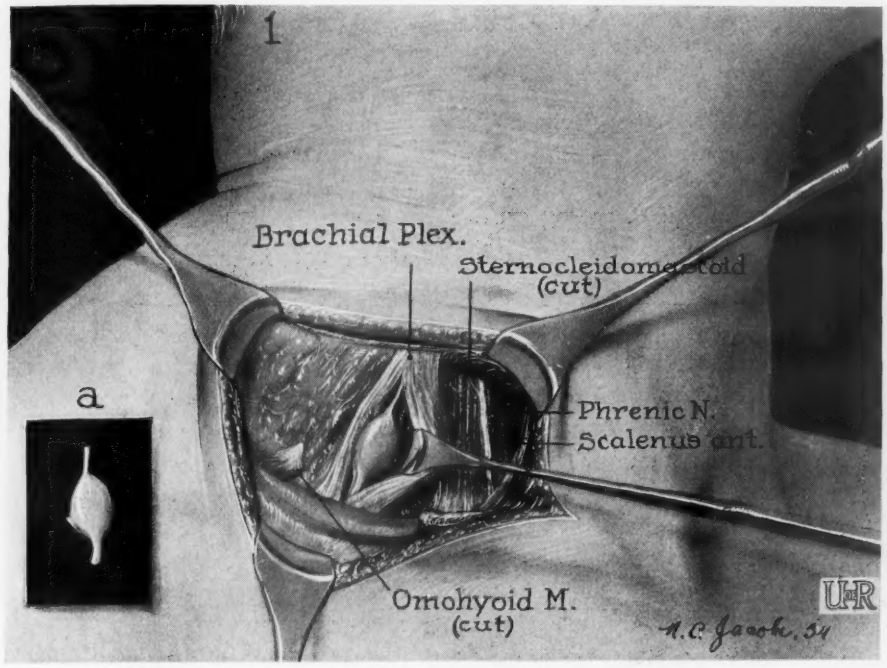

FIG. 2.—H. W. Operative exposure of neuroma on the posterior cord of the brachial plexus. (*a*) Neuroma after excision.

Operation.—On exploration of the region through an anterior approach it was found that the transverse process lifted the scalenus medius muscle directly under the brachial plexus. On one of the posterior cords of the plexus an elliptical mass was felt directly over

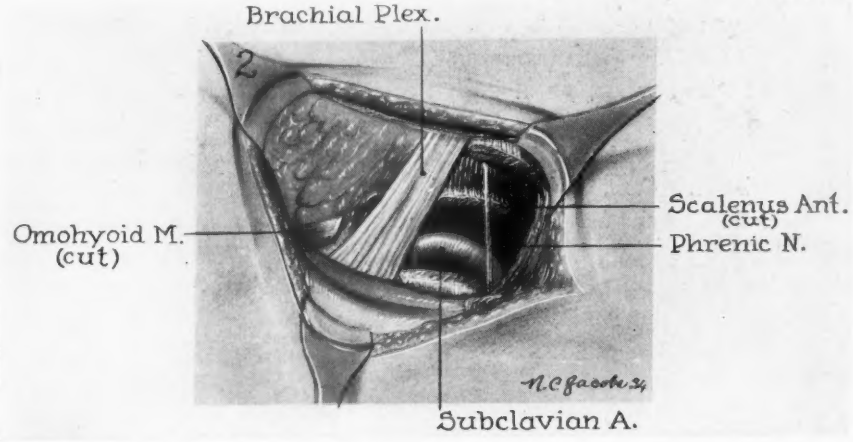

FIG. 3.—H. W. After removal of the neuroma the anterior scalene muscle is divided as in the operation for cervical rib, in order to allow forward displacement of the subclavian artery and brachial plexus.

the transverse process. On gently separating the anterior cord of the brachial plexus, it was found that this mass was a neuroma of the brachial plexus (Fig. 2). This neuroma

was removed (Fig. 2*a*). The scalenus anterior muscle and fascia were divided (Fig. 3) in order to allow the forward displacement of the plexus and subclavian artery. The transverse process was not removed.

Postoperative Course.—The patient's symptoms were relieved at once. She has had no more attacks of tingling, numbness and no more pain in the right hand. She can now stand cold on the right hand without discomfort.

Final Diagnosis.—Vasomotor neurosis associated with a neuroma on a posterior cord of the brachial plexus apparently induced by irritation of this posterior cord by an elongated transverse process.

There are cases on record, for example, where the vasomotor irritation from a cervical rib has resulted in thrombosis of the radial and brachial arteries with maintenance of circulation in the subclavian and axillary arteries.[13] Mechanical pressure effects can in such an instance be entirely excluded. The thrombosis and associated gangrene in one or more digits was due entirely to arterial spasm. This evidence together with that of a similar thrombosis resulting after long periods of frequent angiospastic attacks in Raynaud's disease provides conclusive proof, in my estimation, that continued spasm favors ultimate thrombosis in the spastic vessels, and argues strongly in favor of overcoming such spasm whether its presence results from peripheral nerve irritation, Raynaud's disease, or accompanies organic arterial disease. Aside from distant vascular effects associated with peripheral nerve irritation from pressure or scar tissue involvement, the vasomotor neuroses secondary to other nervous disorders do not ordinarily cause serious symptoms and rarely will require treatment for themselves.

ANGIOSPASM CONSECUTIVE TO TRAUMA

The association of vasomotor disturbances with the late effects of trauma furnishes a fascinating story. There are two main types. The first of these is paroxysmal angiospasm which closely resembles the symptomatology of a local Raynaud's condition with painful ischemic attacks, particularly on exposure to cold.[14] The trauma may be many months in the past and unassociated with any gross scarring. Presumably this is a reflex phenomenon associated with scar tissue involvement of small nerves. We have not found evidence of general decalcification in this form of angiospasm and its treatment is similar to that of Raynaud's disease.

The other form of vasomotor disturbance resulting from trauma is called painful osteoporosis or reflex traumatic arthritis. Some will question the discussion of this interesting and important clinical syndrome among the angiospasms as there is definitely present, at least during certain stages, an increased vascularity about the inflamed joints. Leriche and his coworkers,[15, 16] have ascribed the bone changes entirely to vasodilatation in the bones. However, vasodilatation achieved by removing the vasoconstrictor supply to the part over a period of years fails to produce such bone changes. Furthermore, together with the bone changes, there are usually outspoken evidences of arterial spasm in the cutaneous regions of the distal part of the foot. Consequently it seems to us fitting to consider this interesting sequel

of trauma with the angiospasm which frequently accompanies it. After an injury which may be single or multiple, serious or apparently quite trivial, the individual experiences a period of increasing pain causing disability. Exercise of the part quickly brings on extreme pain after the syndrome is well established. Frequently within a month of the development of symptoms, a marked osteoporosis in the region occurs. This usually differs strikingly from loss of calcium associated with mere disuse in that it gives an early extreme mottling of the bone. The joints usually involved are those of the tarsus or carpus. In the region of the involved joints there is a marked increased vascularity. Frequently there is also evidence of sympathetic stimulation such as excessive perspiration, coolness and clamminess of the anterior part of the extremity, *etc.* We are still in doubt about the exact cause of the bone changes and of the pain. But we know that both of these may remain with little improvement over intervals of several months or even years, although the ultimate result is usually some recalcification and diminution in pain. When the interval has been many months, it is often found that the cartilages between the small bones have been destroyed and ankylosis has occurred. Frequently at the height of the osteoporosis a diagnosis of tuberculous arthritis has been made and amputation has often been advised. However, careful analysis of the bone changes can usually differentiate fairly readily between these two conditions, the chief confusing factor being the protracted course of each. There is a true inflammatory reaction in the joint itself but an infectious etiology can be dismissed. Apparently the mechanism of the condition is on a vascular basis. Whether the signs of sympathetic stimulation and arterial spasm are due only to the pain which dominates the clinical picture or whether there is a true vasomotor imbalance with vasodilatation in certain areas as about the joints and vasoconstriction in others, such as distal cutaneous ones, is not known. We sorely need further elucidation concerning the mechanism of the vasomotor changes. In accordance with our uncertainty concerning the mechanism causing the syndrome, the treatment at present is empirical and its rationale is uncertain. However, the results in certain cases have been very gratifying. Leriche first advised periarterial sympathectomy. Although he and his school are still enthusiastic supporters of this form of treatment, it seems that even in their hands it has probably not proved thoroughly satisfactory as they have more recently resorted in a number of instances to ganglionectomy. It has been our experience that periarterial sympathectomy usually results in improvement but most frequently this improvement is only temporary. Sympathetic ganglionectomy we have felt to be more satisfactory and we have achieved temporary relief of pain by anesthetizing the lumbar sympathetic cord. In lesser degrees of the condition, conservatism is justifiable as usually such cases eventually recover fairly completely. If some simple conservative treatment would permanently relieve these cases it would be very useful.

I am convinced that some reflex disturbance in the blood supply of the involved part is a common accompaniment of trauma and that the study of such minor conditions will result in valuable information being obtained, and almost surely in a better understanding of this significant type of reaction. I would also like to suggest that this component of spasm which we have seen playing such an important rôle at times in organic peripheral disease, following trauma, and also as a complication of certain nervous disorders, may possibly prove equally important in certain of the chronic parenchymatous diseases. Here also, it may well pay us to consider not merely the structural changes but the functional condition in the local circulation.

CONCLUSIONS

The existence of arterial spasm has long been known to occur as a comparatively rare clinical entity. Its occurrence in the common arterial diseases of the extremities has only recently been studied. The extreme forms of angiospasm may undoubtedly lead to thrombosis and gangrene. Vasomotor disturbances following trauma present an interesting though complicated group of conditions which in their marked forms occur as striking clinical pictures and in their milder forms as frequent disturbances.

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INDICATIONS FOR AMPUTATION IN PROGRESSIVE ARTERIAL OBLITERATION OF THE LOWER EXTREMITIES*

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HOWEVER much we may dislike to amputate a limb for arterial deficiency, we must still accept the end-result of a process in which the rate of obliteration of arterial supply exceeds that of the development of collateral vessels, as amenable only to surgical removal. Moreover, an early understanding of the symptoms and physical signs which render a continuation of conservative treatment either unsafe or useless will not only return the patient to a state of physical and mental comfort but, in most cases, will relieve an economic burden which patient and hospital can ill afford to maintain.

The indications for and the level at which amputation should be done are not always clear cut. Increasing experience in the care of these patients within the hospital and a careful follow up study of results after treatment will to a striking degree clarify the indications for a given operation. This is particularly true in the group with senile gangrene and in the group of patients with gangrene associated with diabetes mellitus.

It is my privilege tonight to discuss that group of patients suffering from thrombo-angiitis obliterans, senile gangrene, and the so called diabetic gangrene, beginning at the point where conservative measures have failed. I shall try to outline as clearly as I can those indications for which we look in an effort to determine when and at what level amputation can be done.

By conservative measures, I include all procedures except amputation of a digit or limb. For the purpose of this discussion, then, an operative procedure as extensive as a lumbar ganglionectomy is included as falling within the realm of conservative measures. Without going into a detailed discussion of the various methods advocated for the care of these patients, it would seem necessary to briefly summarize the measures which have been found of practical importance to us in the treatment of these cases in the Peripheral Vascular Clinic at the Massachusetts General Hospital and in the Diabetic Clinic at the New England Deaconess Hospital. It is not the purpose of this paper to give reasons why certain procedures are or are not used by us. It would seem that we might render a more clear cut conception of these conditions as we see and treat them, if we avoid an academic discussion of the various methods advocated and present only an outline of the procedures which have seemed to us to be most useful and practical. From this background, we shall attempt to outline the findings from which we determine that conservative methods have failed or are not worthy of a trial. Let me

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point out that, although the management of the three groups of cases is similar, the indications for a major amputation and the factors involved in the selection of treatment in each group are distinctly different. The man whose experience has been largely confined to the treatment of thrombo-angiitis will have many disappointments and a high mortality in his early experience in the management of diabetic gangrene. Conversely, the man whose experience has been largely with diabetic gangrene may sacrifice an unnecessary number of extremities in the group of patients with thrombo-angiitis obliterans. I need not warn you that as our experience increases in the care of these patients, we more clearly visualize the course of the process in each individual case; a given foot which in our early experience might seem hopeless may later be recognized as amenable to a conservative operation.

RÉSUMÉ OF CONSERVATIVE MEASURES

It is the aim of all conservative procedures to: (1) control infection; (2) relieve pain; (3) stimulate the development of a collateral circulation (it is quite possible that the improvement which takes place in many of these patients is due to a readjustment of the circulatory needs of a given foot rather than to an actual increase in collateral vessels); and (4) release vasomotor spasm. These results we have tried to obtain by:

(1) Rest in bed with sufficient medication to control pain. It is important that the resting position of the involved foot is such that the foot is not blanched (Buerger's angle of circulatory sufficiency).

(2) Large hot packs of saturated boric solution used to control a spreading infection.

(3) Local applications of Dakin's solution during the day, occasionally Obtundia (Clapp's) for a painful ulceration, or a small amount of boric ointment at night. At the Deaconess Hospital, hexylresorcinol solution S T 37 (1-4) is used when a nonirritating moist dressing is desired.

(4) Careful hygiene of both feet; avoidance of pressure areas by placing a small pillow just above the heels; daily massage with hydrous lanolin, Balkan frame and, in diabetic patients, particular care to the back.

(5) Buerger's postural exercises[1] as modified by Allen[2] in all cases except those with evidence of lymphangitis or spreading infection.

(6) General supportive measures such as iron for anemia, a well-balanced diet, and a high fluid intake.

(7) Smoking is advised against. Since the appearance of Maddock and Coller's[3] work, stringent efforts are made to prohibit the use of tobacco by patients with thrombo-angiitis obliterans; less attention is given to it in the arteriosclerotics with or without diabetes.

(8) Peripheral sensory nerve block, first suggested by Silbert[4] and developed by Smithwick and White,[5] is our most important agent in the management of the more painful lesions in the lower extremities of patients with thrombo-angiitis obliterans; it is of occasional benefit in arteriosclerotic gan-

grene, but is only rarely if ever indicated in the presence of diabetes mellitus. It not only gives complete relief from pain but combines with this the vasodilating effect of a sympathetic ganglionectomy.

(9) Protein shock[6] using typhoid vaccine intravenously is much less frequently employed than in the past. It gives a temporary vasodilatation, is of value in the study of the vasomotor index;[7] and in those cases without sufficient discomfort to warrant nerve block it shortens the hospital stay and at times is very helpful in obtaining a temporary vasodilatation in preparation for and following amputation of a digit. It is dangerous in arteriosclerotic and diabetic gangrene[8] because of the possible thrombosis which may occur during the prodromal stage when the rate of blood flow is diminished. It may prove particularly valuable in a group of thrombo-angiitis obliterans patients when the upper extremities are involved.

(10) Sympathetic ganglionectomy has been done in only three of the 53 cases of thrombo-angiitis obliterans and in none of the cases of either senile or diabetic gangrene in this series.

(11) Alternate negative and positive pressures[9, 10] ("Pavaex" treatment of Herrmann and Reid[11]) have not been used over a sufficiently long period of time to have influenced the group of patients studied in this series.

METHOD OF EXAMINATION

We depend upon a careful clinical examination for the determination of the arterial supply to a given extremity. Both legs are bared to above the knees.

Inspection.—Rapid blanching on elevation and the slowly developing rubor or cyanosis when dependent is one of the most striking and constant findings of a poor arterial supply. The rapidity of blanching, the level to which the rubor extends, the presence or absence of atrophy of the calf or foot muscles, or of a puffy edematous appearance of the foot and toes are carefully noted. Migrating phlebitis, if present, is of great significance.

Palpation of the dorsalis pedis, anterior tibial, posterior tibial, popliteal and femoral arteries to determine the presence or absence of pulsation is of fundamental importance. Temperature variations to touch at the same level on the two sides and at different levels on the same side are important. Whether the foot is moist or dry may suggest the presence or absence of a vasomotor imbalance.

The *oscillometer* is occasionally used but has not been found to be of great clinical value to us.

Roentgenographic examination of the vessels to determine the presence or absence of calcification may be of value in differentiating between thrombo-angiitis obliterans and arteriosclerosis. It is of no value in determining the level at which amputation if necessary should be done.

Spinal anesthesia,[12] novocaine block of one or more peripheral sensory

nerves[13] or typhoid vaccine intravenously[14] may be used to determine the degree of vasomotor spasm present.

THROMBO-ANGIITIS OBLITERANS

Diagnosis.—A diagnosis of thrombo-angiitis obliterans is made in a man, usually young (the average age at onset of 53 patients was 41 years), with symptoms of years' rather than months' duration, most frequently beginning as cramps in the calves of his legs, or a soreness in his arches after walking. Occasionally one sees a rapid, fulminating type of disease with a short history and rapidly spreading gangrene. On entrance to the hospital, there will usually be a painful open lesion on one or more toes of a swollen dusky foot with the usual signs of arterial insufficiency. The disease may involve all four extremities, nearly always involving the lower extremities first, usually one at a time. Migrating phlebitis, if present, is almost pathognomonic. Roentgenograms, except in the occasional patient where there is a superimposed arteriosclerosis, will be negative for calcium deposits in the arteries. Usually the vasomotor index is low but occasionally quite marked. Collateral circulation may be developed to an extraordinary degree.

Incidence of and Indications for Operation.—Most patients who seek hospital treatment for thrombo-angiitis obliterans have a painful open lesion. This usually involves one or more digits of a foot and as the disease progresses other extremities are involved. A high percentage of cases who come for hospital treatment will have had some type of amputation. Of 53 cases entering the Massachusetts General Hospital during the past seven years, 32 per cent have had a minor amputation and 26.4 per cent (Table I) have had an amputation of a lower extremity.

TABLE I

THE INCIDENCE OF MINOR AND MAJOR AMPUTATIONS IN THROMBO-ANGIITIS OBLITERANS, ARTERIOSCLEROTIC, AND DIABETIC GANGRENE

Diagnosis	Total Cases	No Op.	Minor Amp.	Major Amp.
Thrombo-angiitis obliterans	53	41.6%	32.0%	26.4%
Arteriosclerotic	55	36.4%	9.1%	54.5%
Diabetic	253	51.0%	2.7%	46.3%

In the management of these cases it is exceedingly important to recognize: (1) the marked tendency for the development of collateral circulation, and (2) the resistance which this group of patients has to the development of a septicemia or a rapidly progressive infection. This is in striking contrast to the deaths from septicemia or from local infections in any group of cases with gangrene and diabetes. In no instance have we had a death from septicemia in this group of patients, and in only four cases did a rapidly ascending infection or progressive gangrene necessitate a guillotine amputation.

The slowly progressive course of the disease, the frequent involvement of all extremities, together with the remarkable development of collateral circulation which occurs, make it safe and, we feel, important to avoid a major

amputation even at the expense of prolonged hospital treatment. For this reason, except in rare instances, a major amputation is not done without first attempting the removal of one or more digits and, if necessary, drainage of the deeper structures of the foot. Pain is no longer an indication for major amputation. Sensory nerve block with its accompanying vasodilatation and relief from pain has been the greatest single factor in lowering the incidence of major amputations. Smithwick and White[15] report 25 cases with and without nerve block showing a drop of from 74 per cent to 30 per cent in the number of cases which come to a higher operation.

An early high operation may be necessary for (1) progressive extension of gangrene in spite of conservative measures; (2) rapidly ascending infection; and (3) destruction of so much of a foot that insufficient structures remain to insure adequate weight bearing should complete healing take place. The Gritti-Stokes amputation is the procedure of choice (Table II). Rarely is a higher amputation necessary. We do only an occasional amputation below the knee in these patients. When infection or gangrene is extending rapidly a guillotine amputation may be indicated. This is done below the knee if circulation will permit, otherwise above. In most cases when this operation has been done below the knee a closed amputation, usually a Gritti-Stokes, is advised as soon as the patient's general and local conditions will permit.

TABLE II

INCIDENCE OF AMPUTATIONS AT VARIOUS LEVELS

	T. A. O.	Arteriosclerotic	Diabetic
Total cases operated	31	35	124
Amputation of toe	54.9%	14.3%	5.6%
Guillotine	9.7%	8.6%	6.4%
Lower leg	6.4%	2.8%	12.1%
Gritti-Stokes	25.8%	22.8%	15.3%
Thigh	3.0%	51.4%	60.5%

ARTERIOSCLEROTIC GANGRENE

Diagnosis.—Arteriosclerotic gangrene is usually present in older people (the average age at onset of 55 patients was 65 years), affects both men and women, and in most cases one leg at a time. The upper extremities are rarely involved except by sudden massive thrombosis or an embolism. There is nearly always calcification of the vessels, very little vasomotor influence and only a fair tendency to develop collateral circulation. Calcification of the vessels of the foot and leg is usually demonstrable roentgenologically. There is a normal blood sugar.

Incidence of and Indications for Operation.—Patients with senile gangrene seek relief for three reasons: (1) an active lesion such as a small painful ulceration or slight infection around a nail; (2) gangrene of one or more toes; and (3) severe pain without any open lesion. Unless actual gangrene involving the deeper structures of a digit exists most patients respond to the hospital routine already outlined. Undoubtedly the early response

to treatment represents a readjustment of the circulatory needs of the extremity rather than a new development of collateral circulation. Many of these patients come to the hospital with severe, unbearable rest pain to become pain free within two weeks' time without treatment other than the simple measures previously described. Gradual rehabilitation by regular graded, active exercises, careful foot hygiene, and living within circulatory possibilities may postpone actual gangrene for years.

Amputation of a Toe.—In only five or 9.1 per cent of 55 cases of arteriosclerotic gangrene was amputation of a toe successfully accomplished (Tables I and II). It is usually safe and may be successful providing that there is no lymphangitis for: (1) osteomyelitis of a phalanx without actual gangrene; (2) localized gangrene of the tip of a digit if there is pulsation in the popliteal artery and no definite level of temperature or color change in the lower leg, providing the patient is not having severe pain after two weeks of hospital treatment; and (3) in the presence of good pulsation in the dorsalis pedis artery. Major amputation was done in 54.5 per cent of our 55 cases. It is usually indicated for: (1) severe pain with or without actual gangrene which is not controlled after three weeks of careful hospital treatment as outlined above; (2) gangrene of one or more digits without pulsation of the dorsalis pedis artery, with deep infection or lymphangitis not controlled by constant moist dressings; and (3) gangrene of all or a part of a digit with or without pulsation in the popliteal artery when there is a definite level of temperature or color change with the foot in the dependent position.

Amputation through the upper third of the lower leg may be safely and usually successfully done in a patient under 70 years of age who is in good general condition, and has good pulsation in the popliteal artery providing that the skin at the level of incision is in good condition and there is no temperature or color change, and no lymphangitis above the level of the ankle.

A Gritti-Stokes amputation commands about the same requirements as an amputation through the lower leg, except that good collateral circulation around the knee is of more advantage than pulsation of the main vessel. This would be shown by absence of pulsation in the popliteal artery and no temperature or color change above the ankle, when the foot is dependent. Neither a lower leg nor a Gritti-Stokes amputation should be done if the patient's general condition is poor and there is little likelihood of his using an artificial limb.

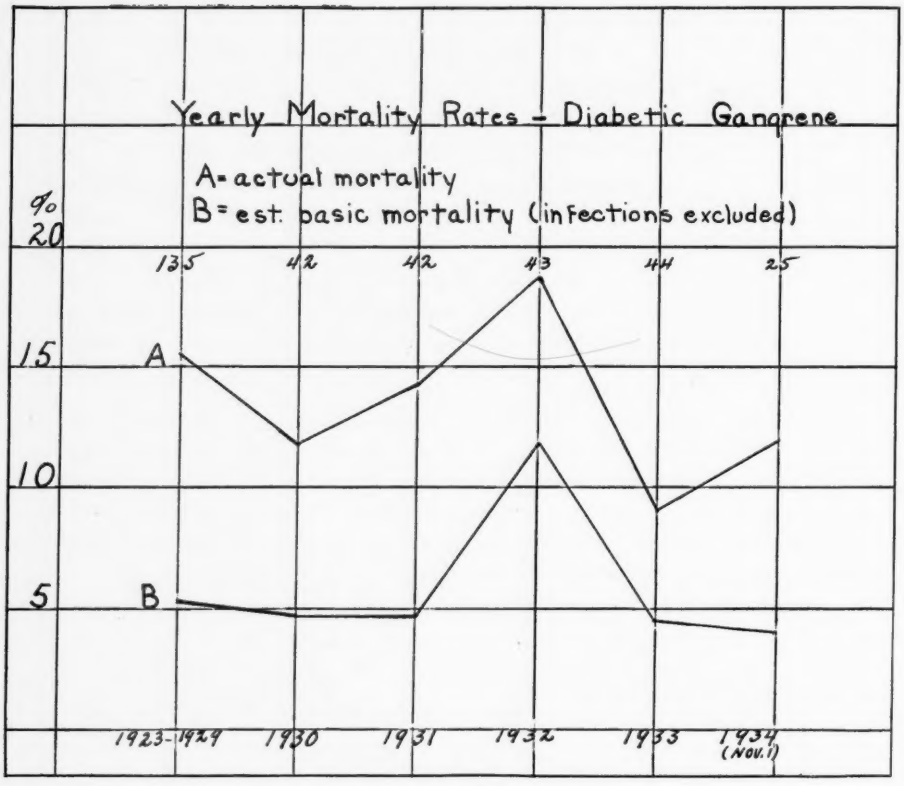
A low or midthigh amputation is the simplest and safest amputation. It should be done on most patients over 70 years of age, when the patient's general condition is poor or when a previous accident such as cerebral hemorrhage or blindness makes the use of an artificial limb impossible.

DIABETIC GANGRENE

Diabetic gangrene appears more frequently at an earlier age (the average of 312 patients was 64 years) than the arteriosclerotic. There is glycosuria

and elevation of blood sugar. A story of intermittent claudication is more commonly obtained than in arteriosclerotic gangrene, and there is more tendency to develop a collateral circulation; in fact, many patients with diabetes develop an adequate collateral circulation without ever having consulted their medical adviser because of symptoms. We have never seen the combination of thrombo-angiitis obliterans and diabetes.

Gangrene of the arteriosclerotic type in a patient with diabetes mellitus is a wholly different problem from that due to thrombo-angiitis obliterans or



GRAPH 1.—Curve A represents the actual mortality following amputations, minor and major, for diabetic gangrene at the New England Deaconess Hospital. Curve B represents the mortality after deaths due to infection have been excluded. The resulting mortality is largely that of the general arteriosclerotic processes.

even uncomplicated arteriosclerosis. The combination of devitalized tissue from lack of arterial supply and the lowered general and local resistance to infection in diabetes mellitus renders much more uncertain not only the future of the part in question, but the life of the patient. There is no warning of the impending septicemia which frequently ensues. The risk of reamputation is great. The reward of an early and accurate determination of when and where to amputate is short hospitalization, early healing without complications, and lower mortality. The penalty of delay or unwise selection of operation is infection in the stump, long convalescence, reamputation, and

a higher mortality. In the group of diabetic patients at the Deaconess Hospital who have come in with failing arterial circulation and lesions demanding hospitalization, 49 per cent of 253 patients during the past three years have required some type of amputation, and of these only 5.6 per cent had a successful amputation of a digit (Tables I and II).

Graph 1 represents a study of the deaths occurring in these patients at the New England Deaconess Hospital from January, 1923, to November 1, 1934. The general arteriosclerotic processes of the diabetic patient carry with them a definite unavoidable mortality. It is interesting to note from Graph 1 that it approximates 5 per cent, and that with the exception of 1932 this rate has been almost constant. The difference between this and the actual mortality of about 15 per cent undoubtedly represents the mortality either of delayed or inadvisable treatment; in other words, the mortality where infection has made the difference between success and failure.

The indications for amputation in this group are not difficult to interpret if one has constantly before him: (1) the diabetic patient is frequently as old as his age in years plus the duration of his diabetes; (2) beginning gangrene in a patient with diabetes mellitus usually accompanies serious disease of the coronary and cerebral vessels, is frequently associated with failing eyes and heart, and in most cases is the beginning of a big economic load and a relatively short life expectancy; (3) treatment by trial and error is exceedingly dangerous and usually unsuccessful; (4) prolonged sepsis or pain seriously damages the already poor heart and kidneys of these patients and undermines their morale; (5) once gangrene has started the tendency to develop septicemia is greater than the tendency to develop adequate collateral circulation; (6) if a patient comes in for pain and is unrelieved by two or three weeks of conservative hospital treatment, as previously outlined, gangrene if not actually present will eventually develop and early operation is preferable to continued pain and disability, complete loss of morale and finally amputation or death; and (7) the difference between the usefulness of a supracondylar and Gritti-Stokes stump in the diabetic over 65 is not sufficient to compensate for the increased risk of the latter.

The circulatory requirements for specific operations are not materially different from those already described under senile gangrene, except that one must constantly have in mind the increased risk which the diabetic patient runs from infection and from reamputation.

The guillotine amputation, usually in the upper third of the lower leg, is most useful in patients with diabetes (Table II). It is indicated: (1) in the presence of a bacteremia or septicemia; (2) for gangrene with extensive lymphangitis which does not respond to constant hot boric packs; and (3) for a patient with gangrene and extensive local infection, with severe untreated diabetes. This type of patient may be in such poor general condition that early removal of a large area of infection is indicated and primary suture too uncertain.

SUMMARY

The conservative measures used in the treatment of thrombo-angiitis obliterans, arteriosclerotic and diabetic gangrene in the Peripheral Vascular Clinic at the Massachusetts General Hospital and in the Diabetic Clinic at the New England Deaconess Hospital have been outlined.

The incidence of minor and major amputations in 53 cases of thrombo-angiitis obliterans, 55 cases of arteriosclerotic and 253 cases of diabetic gangrene is given.

The indications for minor and major amputations in the three groups are discussed.

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RELATIONSHIP OF THE PARATHYROID GLAND TO CALCIUM METABOLISM*

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The parathyroid glands first made their appearance on the anatomic horizon about 50 years ago, Sandström being the first to recognize them as independent organs. For several years they were considered to be aberrant thyroid tissue, but in the late years of the past century the individual function of the parathyroids was finally recognized, and since that time these glands and the rôle played by their secretion in body metabolism have been of vital interest to the clinician, the research worker, and the biologic chemist. Since these glands have been classed among the organs of internal secretion, the amount of literature on the subject has been enormous. One paper on experimental work alone gives 2,600 references and another well over 500. The various theories as well as the case reports have been, to the clinician at least, most confusing. While the last word on this subject is far from being said, certain fundamentals seem to be established and this paper is an attempt on the author's part to clarify for himself the principles of calcium metabolism and the effect of parathyroid secretions on this metabolism and also to work out a practical method of diagnosis and treatment of the cases in which hyperparathyroid function occurs.

In this paper the citation of cases seen at the Orthopaedic and the Graduate Hospitals and direct references to published papers have been deliberately eliminated and the statements presented are only the personal conclusions drawn from these sources. The publication in the near future of the researches of Doctor White and Doctor Stein will give in more detail much of the data on which these conclusions are based.

One sure fact that seems to be beyond dispute is that the parathyroid hormones play their most important clinical rôle in the deposition and extraction of calcium salts in the body skeleton. To understand the pathologic problems a knowledge of the normal mechanism of calcium deposit is necessary. Bones owe their rigidity to the precipitation in them of calcium phosphate and calcium carbonate, the phosphate being by far the more important. The exact formula of this phosphatic deposit is still in question but is of no consequence to the clinician. Although the experimental data on the subject are still incomplete and often very confusing, it now seems to be a well-established fact that our old theories of the specific power of the osteoblastic cells to lay down new bone must be discarded. According to the present theories the precipitation of calcium phosphate in bone is brought about by the local chemical action of an enzyme, called by Kay and Robison phospho-

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tase, acting on the calcium and phosphorus ions found in normal body fluids that bathe the bones. All body fluids are identical as far as calcium and phosphorus are concerned and blood serum can be considered as an example. This phosphotase is probably supplied by the cells of peri- and endosteum and it is supposed to act by causing a local overproduction of phosphate. It is also supposed to become active only in the presence of another enzyme known as vitamin D.

The calcium and phosphorus ions of the blood serum are, therefore, the raw materials from which the calcium phosphate of the bone is formed. From a practical standpoint, blood serum may be considered to be a supersaturated solution of calcium and phosphorus ions. Its calcium ions can be divided into three distinct fractions. First, calcium that is normally soluble in any solution of the same composition as serum, the amount being about 2 mg. per 100 cc. of blood. Second, calcium held in solution by the parathyroid hormones present in the serum, 4 mg. per 100 cc., and third, calcium combined with protein, 4 mg. per 100 cc., making a normal total calcium content of 10 mg. The first is known as chemically dissolved calcium, and the second as biologically dissolved calcium, and the third as calcium proteinate. The first two forms are in equilibrium with phosphorus and are active in bone formation. The third form is inert and plays no part in calcification. In other words, chemically and biologically dissolved calcium are the active reservoirs of the materials for bone formation. The chemically dissolved calcium is a constant fraction but the amount of biologically dissolved calcium seems to depend on the amount of parathyroid hormone present at any given time in the blood stream.

It seems to be a primary law of body metabolism that the blood serum must always be in this state of supersaturation and that anything that destroys this condition will draw calcium or phosphorus from some other source to maintain it. To put it in another way—the amount of calcium and phosphorus in the blood stream varies with the amount of parathyroid hormone, and this hormone must be satisfied. Normally, this condition is fulfilled by the salts absorbed from the alimentary tract, but if these be deficient, the calcium and phosphorus stored in the bones can be redissolved to make up the necessary amount.

Vitamin D is another factor in maintaining the calcium-phosphorus balance in the blood. This substance is supposed to aid in the absorption of the salts from the alimentary canal in addition to its action on the phosphotase in the bone. Normally, the ingestion of these salts amounts to about one Gm. of calcium and two Gm. of phosphorus per day and a like amount is excreted, 90 per cent of the excretion taking place through the feces and 10 per cent through the urine.

The evidence now at hand as to calcium metabolism can, therefore, be summarized by these statements. First, the bone building mechanisms depend on the blood serum for their raw materials. Second, in normal blood serum there is an approximately fixed ratio between calcium, phosphorus,

parathyroid hormones and vitamin D, the normal ratio between calcium and phosphorus in the adult being about 10 to 4, and in the child about 10 to 5. Third, that any disturbance of this ratio demands an extra supply of calcium or phosphorus from some source to reestablish this ratio. Fourth, if this extra supply can be obtained from ingested salts no bone changes will appear, but if it cannot be so obtained, the reservoirs of calcium and phosphorus in the bone are called upon to supply the deficiency and decalcification makes its appearance. To state the same fact in another way, if one of the factors of this serum ratio be changed, all must be equally changed to prevent bone disturbance.

In addition to its bone forming functions, calcium together with other ions is supposed to maintain the physicochemical equilibrium necessary for normal irritability of muscles and nerves. Its especial function is to lessen irritability at the neuromuscular junctions both in the heart and in voluntary and involuntary muscle fibers.

In taking up the pathologic changes due to irregularity of parathyroid secretions, the clinical manifestations of lowered parathyroid secretion are now fairly well understood. Lowered parathyroid activity causes a marked diminution in serum calcium, but no loss of the total calcium of the body by excessive secretion, the excess calcium being stored in the spongy bone. The carpopedal spasms, general convulsions, *etc.*, which are the distinguishing signs of tetany, are due, therefore, to the lowering of the total calcium content of the blood by the marked lessening of the biologically dissolved fraction. The introduction of calcium through the alimentary canal, especially when given with a meat free diet, is of distinct use in combating this hypocalcemia and the symptoms caused by it. Of more importance is the use of parathyroid hormone. In 1924 this hormone was first extracted by Collip and has been used both experimentally and clinically. Its intramuscular injection is followed by a rapid rise in serum calcium and an increased excretion of both calcium and phosphorus with diminution of the symptoms of tetany.

The effect of hypersecretion still presents many unsolved problems and the facts presented here are drawn mainly from the experimental and clinical work done at the Orthopaedic Hospital originally by Doctor E. P. Corson White and recently by Doctors White and Stein.

The addition of excessive parathyroid hormone to the blood stream brings about an excess of calcium ions and a lowering of phosphorus. At the same time there is a marked increase in the amount of calcium and phosphorus excreted. Normally, as has been said, 90 per cent of the calcium is excreted by the alimentary canal and 10 per cent by the kidneys. In hyperparathyroid conditions this ratio is reversed and up to 90 per cent of calcium is eliminated by the kidneys and as little as 10 per cent by the alimentary canal. If this excessive calcium excretion persists, marked kidney dysfunction occurs and a marked rise in the phosphorus content of the blood is brought about. Therefore, while a high calcium and a low phosphorus value is seen in the

early stages of hyperparathyroidism, the blood picture may be entirely changed by the loss of kidney function. It is, therefore, necessary to examine the eliminating power of the kidneys in all cases in which the blood picture shows an abnormally high per cent of calcium. This extra calcium in the blood is necessarily drawn from the salts stored in the bone structures and decalcification will occur unless the normal blood balance is restored either by eliminating the causal factor or by increasing the ingestion of calcium and phosphorus from the alimentary canal. The blood picture above described is not necessarily caused by overactivity of the parathyroids—other pathologic states such as sarcoma, metastatic carcinoma, acidosis, and at times tuberculosis, will give the same picture—but the history, the clinical examination, and especially, the roentgenographic studies, will go far in clearing up the diagnosis.

The clinical symptoms of hyperparathyroidism are mainly caused by these changes in the blood, and give the clinical picture of generalized osteitis fibrosa first described by Von Recklinghausen in 1891. The serum calcium rises to 12 mg. or over. Serum phosphorus falls to 2.7 mg. or under. The urinary output is increased to the point of polyuria. Urinary and renal calculi may be found. Renal damage occurs. The muscles weaken and become easily fatigued and often painful. The gastro-intestinal tract often shows signs of irritation, leading to nausea, vomiting, or constipation. The long bones progressively decalcify, becoming thinned and distorted. Eventually fractures may occur. Bone and joint pain and inability to bear weight are early and persistent symptoms. These bone changes are fairly well distributed throughout the skeleton. The bone marrow is damaged and fibrous tissue replaces the normal cells. This fibrous change is progressive and gives rise to tumorlike masses within the bones. Cystic changes due to necroses occur, and when the fibrous tissue be invaded with giant cells, giant cell tumors will form. These giant cells can be found also in the walls of the primary cysts. Hemorrhage into these masses will give the typical brown tumors of Von Recklinghausen's disease.

Given a case in which the diagnosis of hyperparathyroidism is established, is there any quantitative test that will accurately estimate the amount of parathyroid secretion present? From an experimental standpoint such a test can be made which is known as the Hamilton test, and can be used clinically to estimate grossly the amount of parathyroid hormone present. However, we can still rely on the clinical picture plus the repeated blood tests in planning treatment.

This treatment is based on the fact that hyperparathyroid function throws into the blood stream an excessive amount of its secretion, thus destroying its normal ratio with calcium, phosphorus and vitamin D. To overcome this effective treatment must either reduce the amount of secretion or utilize the excess. Three types of treatment must be considered—surgery, roentgen ray, irradiation and diet.

Surgery when used alone is distinctly limited in its effectiveness. The

parathyroid glands vary greatly, both as to their number and their position. They can be classed in two groups—those that are developed from the third bronchial cleft, and those developed from the fourth. The first group, usually two in number, lie behind or in the substance of the thyroid gland. The second group lie along the carotids and trachea and may extend well down into the thoracic cavity. Adenomata of these glands may form and when they can be diagnosed their extirpation is, according to some case reports, of distinct value. Removal of one gland will at times give temporary relief, but the remaining glands tend to hyperplasia and secondary removals are often necessary. Reports in literature of the operative results vary so much that no definite statistics can be given. It would seem, however, that surgical extirpation alone has given permanent results in a very small percentage of cases.

Roentgen ray irradiation is still in the experimental stages. It has been shown that the activity of the glands can be temporarily or permanently diminished by the use of fractional doses given over the whole parathyroid area at three- or four-day intervals. The total dosage employed is usually about 80 per cent of the erythema dose. It has the advantage of covering the whole parathyroid bearing area, but has the disadvantage of inhibiting thyroid function at the same time. A few successful cases of its use in cystic bone disease and giant cell tumor have been reported, but the amount of the dosage and permanence of its effect have not as yet been sufficiently tested.

At the present time treatment by means of absorption from the alimentary canal seems to be the best method of combating the effects of parathyroid hyperfunction. To accurately prescribe the needed amount of calcium and phosphorus intake it is necessary to know the exact degree of negative imbalance in each individual case. That is to say, each case must be tested to determine exactly how much the calcium or phosphorus excretion exceeds the intake. Such testing can be done only with hospitalized patients and by trained technicians. The exact amount of calcium phosphorus intake and output must be measured. In the Orthopaedic Hospital this is done for three consecutive days. The number of grains of calcium and phosphorus that must be incorporated in the daily diet of the patient to overcome the excess of elimination over intake can then be estimated. Calcium chloride, calcium lactate and disodium phosphate and calcium biphosphate are the drugs usually used. To these are usually added an adequate amount of vitamin D, usually in the form of viosterol (250 D).

To sum up the treatment, accurate dietary measures to increase the calcium and phosphorus intake to a point that will restore normal ratio between these salts and the parathyroid hormone in the blood seems to offer the best method of combating hyperparathyroid secretion. Surgery and the use of the roentgen ray in properly selected cases may be of great assistance, but so far as we know now should be used as adjuncts to diet rather than as the only method of treatment.

In cases with normal kidney function the high calcium-phosphorus diet works exceedingly well. However, in those cases in which there has been marked kidney damage, operative removal of parathyroid tissue or irradiation together with a very small amount of extra calcium and phosphorus by mouth will probably save the patient from the pathologic calcification so often caused by damaged kidney.

There are at present so many side issues that are still in the controversial state that to discuss them would only befog the main picture. We can, however, hope that as experimental work goes on, and our knowledge increases, a more clearly cut and practical method of handling the whole field of faulty calcium metabolism may appear and that many of the puzzling problems, such as Paget's disease, nonunion or faulty union of fractures, the various types of rickets, osteomalacia and osteogenesis imperfecta may finally yield to an understanding use of surgery and physiologic chemistry.

THE OBER OPERATION FOR SCIATICA*

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CASE REPORT.—R. L., a sailor, aged 43. Admitted to Roosevelt Hospital, December 27, 1934, complaining of pain in the right thigh.

History.—Three months before, after sleeping all night in a cramped position, he was seized by a sharp pain in the right leg and thigh. This was continuous from the region of the right hip down the posterior and lateral sciatic region of the thigh and lateral side of the right leg. The pain was aggravated by walking, stooping, coughing and laughing. He was treated on shipboard by the application of hot irons to the painful area, which gave him some, but only temporary relief.

Physical Examination.—Any attempt to flex the thigh upon the body or to extend the lower leg in relation to the thigh caused acute pain. Pressure over the sciatic notch caused great pain. Pressure along the lateral side of the thigh, roughly over the course of the sciatic nerve, was also painful. Doctor Burschell considered the case one of a sciatic neuritis and thought section of ileotibial band should be tried. Roentgen ray of the lumbosacral spine and pelvis was negative. The blood chemistry and Wassermann were also negative.

Operation January 4, 1935, by Doctors Cave and Amendola. Incision six inches long, extending from the anterior superior spine on the right outward and downward, bisecting a line drawn from the upper edge of the greater trochanter of the right femur to a point directly over the spine of the ischium. The tensor fasciæ latæ was found to be exceedingly taut, particularly just below and lateral to the anterior superior spine. An incision seven and one-half inches long was made dividing the fascia lata well out lateralwards. As this was done the fascia seemed to spring apart like a snapped piano wire. It was then undermined for approximately three inches; this seemed to relieve the tension immediately. Several intermuscular taut fibrous bands were cut across. Wound sutured with silk throughout.

Postoperative Course.—Uneventful, except for the amazing disappearance of the pain six hours after the operation. The only discomfort the patient has felt since the operation is the wound pain. Out of bed on the eleventh day without experiencing any weakness of the thigh muscles in the right leg, or pain of which he previously complained.

Microscopic Examination.—Of the few strands of tissue removed at operation, showing a very few scattered lymphocytes, no inflammatory exudate was present.

COMMENT

DR. FRANK R. OBER, of Boston, who first suggested this operation for the cure of sciatica, has had his paper accepted for publication. It is with his knowledge and consent that I am presenting this patient before this society and describing this procedure of his before the actual publication of his paper. In a personal communication, Doctor Ober tells me he has operated upon 24 patients with sciatica by this method, ten of whom were bilateral. He believes that there are several imporant diagnostic points:

(1) Straight leg raising is usually limited whether sciatica is present or not.

* Presented before the New York Surgical Society, January 23, 1935.

(2) When these patients are asked to sit and bend the body forward, with the legs extended on the examining table, it is very rare for the lumbar spine and pelvis to flex even to right angle. Many of these patients are unable to stoop over and touch the floor with their hands.

(3) In most cases, Ely's sign is present.

(4) The most important diagnostic sign of the contracture is obtained as follows: The patient is placed directly on his side. The examiner places one hand on the pelvis to steady it and grasps the patient's ankle lightly with the other hand, holding the knee flexed at a right angle. The thigh is abducted and extended in the coronal plane of the body. If the contracture described above is present, the leg will remain abducted, the degree of abduction depending on the amount of contracture present. This sign is present both in the conscious and the anesthetised patient. If there is no contracture present, the thigh will abduct beyond the median line.

The treatment of this condition resolves itself into one factor and that is the relief of the contracture. In very severe cases, an operation is performed in the following manner:

(*a*) An incision is made from just below the crest of the ilium down to the tip of the trochanter directly over the contracted iliotibial band.

(*b*) The fascia lata is exposed forward as far as the anterior superior spine and backward to the edge of the gluteus maximus muscle. The area of the greatest contracture of the fascia can be seen readily and felt easily.

(*c*) The fascia is now divided transversely from just below the anterior superior spine to the anterior border of the gluteus maximus muscle. There is immediate separation of the cut edges for a distance of three-fourths to one and one-half inches, depending upon the amount of contracture present. If the operator now attempts to carry out the test described above, it will be shown that the thigh will completely abduct.

Discussion.—Dr. Russel H. Patterson thought we should be very conservative in our discussion of these cases, first, because there seems to be no consistently definite well identified pathology described. So far, operations have been done empirically for the relief of pain more than for the correction of well understood pathologic disease entities. And, secondly, there are so many different types of operations for sciatica: (*a*) Several years ago Dr. Percy Roberts, at the Hospital for Ruptured and Crippled, freed the gluteus medius and gluteus maximus muscles from the crest of the ilium and got good results in his cases. (*b*) For a time there was quite a wave of enthusiasm for injecting saline solution, procaine or alcohol or mixtures of these solutions in and about the sacral canal and sciatic nerve with reports of relief in many cases. (*c*) There is a group in the Mid-West who have been stripping the gluteal fascia off of the crest of the ilium and getting good results. (*d*) Recently Freiberg of Cincinnati has called attention to contractures of the pyriformis muscle causing symptoms of sciatica.

Dr. Donald Gordon did not believe that what we saw and what doctors called sciatica was a sciatic neuritis. The neurologists demanded that there be hyperesthesia, anesthesia, or motor changes to make a diagnosis of true sciatic neuritis. He did not think nature ever shortened the sciatic nerve so that, as has been done, it could be stretched by manipulative measures to

relieve in any way a so-called sciatic neuritis. Doctor Ober's procedure, which he carried out on a case of low back pain at Fifth Avenue Hospital last August, makes it seem quite obvious that no sciatic neuritis with true neurologic changes is going to clear up as easily as that. He has felt for many years that sciatica has been associated with contracture of some sort in various places and believes that Doctor Ober's procedure has a very distinct place in treatment of the sciatic syndrome.

DR. CLAY RAY MURRAY had done three of these cases at Presbyterian Hospital and thought that the operation should be limited to selected cases, insisting on all of the signs Doctor Ober has described. Doctor Ober himself stated not long ago that he had no definite idea of the exact nature of the pathology in these cases and agreed that the procedure was somewhat empirical. This is all the more reason for restricting its use to clearly defined cases.

In one of our cases we carried out some rather experimental procedures, for in many it has been presumed that the relief is as much psychic as physical. One of their patients was a carpenter who had suffered for two years from intermittent sciatica, following injury. He had been unable to sleep without sedatives for two months. He presented all the signs Doctor Ober has enumerated. They made a skin wheal along the line of the intended incision. When asked if this relieved the pain, he said it did not. They then made an incision through the skin, first telling him that they were going to cut. Again they asked if this relieved the pain and the patient answered, "No." The Ober signs were still positive. They next anesthetized the fascia along the line of proposed incision. The pain was unrelieved and the Ober signs continued to be positive. Tension was most marked over the gluteus maximus fascia, and not so marked over the fascia of the tensor fasciae femoris. The posterior gluteal fascia was then incised without relief. When, however, they went through the fascia over the outer portion of gluteus, it separated suddenly and widely, and the patient exclaimed, "I felt that all down my leg! Now, my pain is gone!" An immediate test showed the Ober signs now negative. This was the first time the patient had been without pain for two months. Obviously, there was no psychic element in this pain or in its relief.

While it is true that no definite pathology has been described to explain this sciatic pain, in a patient with real sciatica and showing the required signs it is an act of mercy to perform this operation. It can be done under local anesthesia and permits the patient to be out of bed six days or less after the procedure.

DOCTOR CAVE.—It is much more advisable to carry out the procedure under local anesthesia as more information may be obtained in that way. I understand that Doctor Ober has now operated upon 24 cases, ten of which were bilateral. In a personal communication, he has stated that for the most part relief has been almost immediate and satisfactory, though in a few instances the results have come on slowly.

OBSTRUCTIVE JAUNDICE

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Clinical obstructive jaundice patients have a visible discoloration of the skin, mucous membranes, and sclera. Chemically, they have a retention and accumulation of bilirubin in the blood stream. The normal content circulating in the blood varies from one part of bilirubin to from 400,000 to 600,000 parts of blood serum. When the concentration reaches from 1 to 40,000 the skin becomes tinged and clinical jaundice appears. Icteric index is the measure of intensity of the color of the blood serum compared with a standard. The normal icteric index varies from three to six. The clinical evidences of jaundice are usually obtained when it reaches 20 or more. Between six and twenty is the latent period. Kupffer's cells in the liver and the reticular cells of the spleen are concerned in the breaking down of the hemoglobin into bilirubin. Jaundice arises from the obstruction of the outlet of the bile capillaries after the changed bilirubin has passed through the polygonal cells of the liver plus the bile salts and consequent obstruction and absorption. Obstructive hepatic jaundice presupposes a normal bile formation and a normal mechanical excretion through the apparently normal liver cells. This mechanism does not continue for any great length of time after obstruction has occurred, for normal bile is no longer secreted nor are the liver cells intact. The jaundice, in the beginning, becomes obstructive due to a mechanical factor. This does not remain long, for as soon as the obstruction raises the hydrostatic pressure of the fluid within the ducts to a degree equal to that of the secretory liver pressure, there must, of necessity, be a cessation of flow or even a reversal of direction of the bile current with a function conducive to intrinsic damage to the liver cells. Bile salts are the specific products of the liver parenchyma. The production of bilirubin, together with bile salts, is a function limited to the liver. No other organ can simultaneously produce these two chemical substances. Along with bilirubin, bile acids are always present in normal blood. With the Szilard method, the bile acids of normal blood vary from five to 12 mg. with an average of seven mg. Katayama, by a modification of this method, failed to find bile acid in normal urine. An abnormally rapid rate of hemolysis elevates the bilirubin of the blood serum but does not influence the concentration of bile acids. On the other hand, the production of liver damage increases the bile acids. But this has relatively little effect upon the bilirubin of the blood serum. Even in those cases of cholecystitis with transient biliary obstruction, there is a very definite increase in bile acids of the blood serum. The concentration of bile acids is also greater in some cases with normal icterus indices than in others with hyperbilirubinemia. Therefore, it is of paramount importance to determine

the bile acids in the blood, in addition to bilirubin, since the increase of bile acids is an absolute indicator of liver injury. Urobilinuria is also an index of the extent of the impairment of liver function. Normally, it does not exceed 30 mg. in 24 hours by the method of Elman and McMaster. But in obstructice jaundice, urobilin is negative and excreted in minute amounts forming a striking contrast to catarrhal jaundice. On the other hand, in obstructive jaundice, there is an increased elevation of the icterus index disclosing a hyperbilirubinemia. The bile acids are increased above normal but they do not parallel the icterus index. There is also a distinctive change in the chemical composition of the blood in obstructive jaundice as shown by an increase of cholesterol which is present in two forms: (*a*) free alcohol, and (*b*) combined with acid as esters. Normal blood contains from 160 to 200 mg. per 100 ml. In obstructive jaundice, it is increased and may even reach 1,000 mg. per 100 ml. The icterus index, in these cases, may go as high as 200, bile acids to 75 mg. per 100 ml., and direct and indirect van den Bergh four plus. In making an estimate of the extent of the impairment of liver function, the nonprotein nitrogen, urea, uric acid, and the amino acid nitrogen, therefore, for an adequate estimation of complete liver function, and differentiation of the types of jaundice, biochemical analyses must include icterus index, bile acids, blood sedimentation test, cholesterol, nonprotein nitrogen, urea and amino nitrogen, uric acid of the blood, and urobilin excretion in the urine.

Blood sugar determinations are essential as the carbohydrate metabolism is also disturbed by virtue of the fact that the liver regulates, quantitatively, the amount of carbohydrate that is furnished to the body cells in the varying conditions of carbohydrate intake and bodily activity. The internist should order insulin in the suitable cases.

Since over 50 per cent of the fatal cases are due to postoperative hemorrhage, the clotting time is a very essential test, as the calcium of the blood unites with the bile salts and pigments in an attempt to detoxify the toxic state. In many of these cases, there is a hypochlorhydria as is evidenced by the blood determination of the chlorides. Inflammatory infiltration about the ducts and even about the finer bile channels within the hepatic lobule, along with exfoliated epithelium and thrombic formation within the ducts, themselves, are further evidences of the severe injury to the liver.

During the past ten years, two noted changes have taken place, as is evidenced by the referring of jaundice patients to the surgeons by the internists, especially in those cases where the bile salts plus bilirubin are found together in increased or abnormal quantities, showing obstruction. Also, if I may digress, the direct opposite of sending ulcer cases, especially duodenal and marginal, to the internist with the conviction that patient, persistent dieting and medical care will solve many of these cases where operative procedures are constantly failing and are, therefore, discouraging. The different tests enumerated above, along with the routine physical examination including Wassermann, blood count, especially Schilling, cardiogram, and roentgenogram impresses one with the seriousness of these cases and also the importance

of group consultations, particularly as to the advisability of any operative procedure. A surgeon should have a thoroughly adequate experience before he undertakes to operate on these cases. In the long standing toxic jaundice cases, he is entitled to a helpful sympathy since the mortality rate is high.

All operative procedures, without exception, should be done in two stages. The first one is for drainage of the gallbladder when the obstruction is below the entrance of the cystic duct. It is accomplished by using a modification of the Kocher or Courvoisier incisions, making it more longitudinal. After exploring the common duct and determining the point of obstruction, which is below the cystic duct entrance in 90 per cent of the cases, the gallbladder fundus is then brought up to the aperture of the abdominal incision. Following sufficient packing and walling off, this viscus is drained with a trocar and cannula connected with a rubber tube and the edges of the aperture are held taut with Allis clamps. The female portion of a small size Murphy button is then introduced into the opening of the gallbladder. The male end of the button is inserted into one end of a long rubber drainage tube, following which the corresponding parts of the Murphy button are snapped together. Drainage is instituted in this way and suction is applied if necessary. The advantages of the Murphy button are: the absolute hemostasis of the edges around the opening of the gallbladder, its quick disconnection, and a further important function in the secondary stage of operation. Some surgeons prefer a 28 or 30 F. Pezzer catheter. Following the drainage, performed under a local anesthesia, the chemical analyses of the material obtained and a further check-up in the blood and urine chemistry is regularly made. The improvement that some of these cases show after 72 hours of drainage is remarkable.

The consultants, having agreed upon the time for the second stage of operation, then make a Mayo-Robson or Mayo incision after a spinal anesthesia has been given. This incision is closer to the median line than the first one. If the operator finds that he is dealing with a malignancy of the pancreas, he should disconnect the gallbladder fundus, with its contained Murphy button, and use the other end of the button that was inserted in the drainage tube, and after proper sterilization, incise the neighboring first portion of the duodenum, suture in this part of the Murphy button, and connect the two. Drains are then inserted down to Morrison's pouch and to the Murphy button anastomosis.

If the obstruction consists of a large impacted stone in the lower portion of the common duct, as is so often the case, this area should be dealt with by reflecting and retracting the second portion of the duodenum after incising the peritoneum on the right side. This approach is very simple and easy compared with McBurney's transduodenal approach. A T-shaped tube is then inserted. The gallbladder is not disturbed, since we have an actual or potential pancreatitis to combat, and our gallbladder drainage is absolutely essential in this complication.

If, after exploring the cause of the obstruction, we find a complete stenosis

of the lower part of the common duct with repair out of the question, we then use the proximal stump and insert the end into the proximal duodenum. If the end inserted is split into four parts, using longitudinal incisions of half a centimeter, each of these parts can be sutured to the duodenal wall after their insertion by first threading a nontraumatic suture through the edge before insertion. Greater security is given, along with the bile current, by keeping the edges open so that no stenosis can occur.

Even in those cases of common duct stenosis with virtual destruction, a direct anastomosis should be used. This should be possible by hooking a dull retractor, shaped like an aneurysm needle, over the V-shaped juncture of the hepatic ducts to steady our anastomosis. This is especially true on the cadaver in thin subjects where it is comparatively easy to repair the tissues even though a small portion of the common duct is left with which to make an anastomosis between the duodenum and the common duct.

The point that I wish to emphasize is never, under any circumstances, resort to the old and obsolete technic which is exemplified by the Sullivan tube method, in which a space is left filled in by a rubber tube and covered over by various methods. From my observations, they are never successful since the stenosis becomes more acute and the adhesions aggravate the extreme seriousness of further exploratory dissection. Therefore, a direct anastomosis of at least a part of the cut edges of the common duct is essential. Failing to accomplish this, an anastomosis of even a small stump of the upper portion of the common duct to the duodenum should be made. On the cadaver in thin subjects, it offers no great difficulty.

CONCLUSIONS

To improve the clotting time, transfusions of whole blood and glucose solutions should be administered. At the present time, we fully appreciate the extreme value of these solutions in this complication, a fact that was unknown a few years ago. Ten per cent calcium chloride solutions and ampules of calcium agglutinate are also useful, in this condition, since they increase the blood calcium. Chloride deficiency is improved by the introduction of normal saline solutions and diluted hydrochloric acid solutions by mouth. One dram should be given daily in divided doses, gradually increasing the amount. Insulin should be administered in those cases showing increased blood sugar. The diet consists of various fruit juices and cereals, unless contradicted by some special complication.

These patients suffer a fall of body temperature during the operative procedures. This condition has to be counteracted by using hot laparotomy pads, short exposures, and a warm operating room.

The loss of fluids, caused by the drainage, must be counter balanced by the introduction of glucose and saline fluid. After the first 72 hours, the drainage tube can be clamped for a certain time each hour in those cases where it is possible to do so.

The time factor is one of the most serious considerations because, at best,

the patients are poor surgical risks. It is essential to make a thorough check-up to make sure that the drainage tubes, Murphy buttons, and syringes are in perfect condition before operating. This may sound strange to the uninitiated, but the Murphy buttons fit only in pairs and drainage tubes have been known to have a lumen that was imperfect.

There appears to be little or no excuse for the delay in sending these cases to a hospital where a proper work-up and diagnosis can be made. Yet, daily, we see cases admitted in the late stage of jaundice due to obstruction.

SUBCUTANEOUS PERFORATION OF THE JEJUNUM*

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THE number of deaths resulting from trauma to intra-abdominal organs has increased in the past decade. In some of these cases, death has occurred almost immediately, because of extensive injuries to the body, the intra-abdominal lesion forming only a part of the general picture and not, as a rule, the chief cause of death. In a great many instances, however, the patients have lived for many hours after the accident, subsequently dying directly as a result of trauma to the abdominal viscera. It is in these cases that earlier diagnosis and treatment might lessen the high mortality of these injuries. Abdominal injuries naturally separate themselves into two groups: (1) Percutaneous injuries, caused by foreign objects such as bullets, knives, and so forth, in which a penetrating lesion of the abdominal wall is present, and in which the diagnosis of injury to the underlying viscera is made relatively easy, and (2) subcutaneous injuries, caused by a blunt force applied directly to the abdominal wall or even indirectly to the body, as in falling, lifting, or straining. In these injuries, no evidence of trauma to the abdominal wall is present, and the diagnosis and method of treatment in these cases are often exceedingly difficult.

Geill[11] stated that subcutaneous injuries involved the abdominal viscera in the following order: liver, spleen, kidneys, intestines, stomach, bladder, and pancreas; the intestines were involved in 11.1 per cent of his cases.

Intestinal rupture is caused in three ways: (1) By crushing, in which the intestine is caught between some fixed structure such as the spine or pelvis and the direct force is applied perpendicularly to the abdomen; (2) by tearing, in which the force is applied tangentially to the abdominal wall, causing the bowel to be torn from its attachments, and often tearing the mesentery also, and (3) by bursting from increased intra-intestinal pressure exerted by fluid or gas caught in the bowel between angulations of the wall. In TABLE I are listed cases of rupture of the intestines reported by various authors and tabulated according to the situation of the perforation. In some cases, the site of rupture was noted as merely "small intestine" with no specific localization to the duodenum, jejunum or ileum; in other cases, no note was present concerning the situation of the lesion, while in still others, no perforation was found at operation. Allowing for these discrep-

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ancies, it is seen from TABLE I that the small intestine was the site of rupture in more than 90 per cent of the cases, the jejunum and ileum were involved about equally, and together accounted for about 80 per cent, while the duodenum and large intestine were involved in about 10 per cent of the cases. In reviewing the cases reported in the literature, it was noted that the beginning of the jejunum, just distal to the ligament of Treitz and the end of the ileum, in the region of the ileocecal attachment, were by far the most frequent sites of traumatic perforation. This is undoubtedly attributable to the limited mobility of these sections, and their proximity to fixed bony structures, so that conditions conducive to perforation are present when extreme force is applied to the abdomen.

TABLE I

Situation of Subcutaneous Ruptures of the Intestines

Author	Duodenum	Jejunum	Ileum	Cases Small intestine*	Large intestine*	Not noted	Total
Poland[19]	4	14	16	22	5	0	61
Curtis[7]	6	44	38	21	4	3	116
Makins[15]	1	6	7	2	5	0	21
Gage[10]	10	20	43	0	6	6	85
Lund[14]	0	5	6	5	1	2	19
Berry and Guiseppi[3]	26	32	32	25	10	7	132
Tschistosserdorff[25]	1	23	25	0	3	0	52
Battle[1]	32	111	59	2	11	7	221
Stanley[24]	0	3	1	0	0	0	4
Quain[20]	0	4	3	0	5	0	12
Massie[17]	4	14	8	3	1	4	34
Vance,[26] 1923†	3	4	5	0	2	0	12
Rowlands[22]	23	157	158	0	43	0	381
Richter†[21]	0	1	3	4	3	0	9
Vance,[27] 1928	0	3	4	3	0	0	10
Beekman[2]	0	0	2	0	0	0	2
Cooke[4]	3	6	2	0	1	0	12
Totals	113	447	412	87	100	29	1,183

* Situation not given more definitely.

† Multiple ruptures.

Perforation of the intestines of children is not a very common occurrence; the liver is the organ most often injured in abdominal trauma in childhood. Beekman found only two cases among a series of 59 children admitted to the Bellevue Hospital with abdominal injuries. In TABLE II are listed some cases of rupture of the intestine collected from the literature, in which the age of the patient was given as 12 years or less. They are relatively few in contrast to cases of this injury occurring later in life.

TABLE II

Data Concerning Subcutaneous Rupture of Intestines of Patients Twelve Years of Age or Less. The Quantities Given Here Are Included in Those Given in Table IV

Author	Cases	Patients operated on	Died	Operative mortality, per cent	Died without operation	Total mortality, per cent
Poland	3	0	0	00.0	3	100.0
Curtis	2	0	0	00.0	2	100.0
Makins	4	4	4	100.0		100.0
Lund	2	2	1	50.0		50.0
Berry and Guiseppi	24	13	9	61.5	11	83.3
Quain	4	4	1	25.0		25.0
Vance, 1923	2	2	2	100.0		100.0
Massie	8	5	3	60.0	3	75.0
Richter	11	9	3	33.3	2	45.4
Moynihan[18]	9	7	4	57.1	2	66.6
Vance, 1928	2	2	2	100.0		100.0
Beekman	2	2	1	50.0		50.0
White	1	1	0	00.0		00.0
Cooke	3	0	0	00.0	3	100.0
Totals	77	51	30	58.8	26	72.7

CASE REPORT

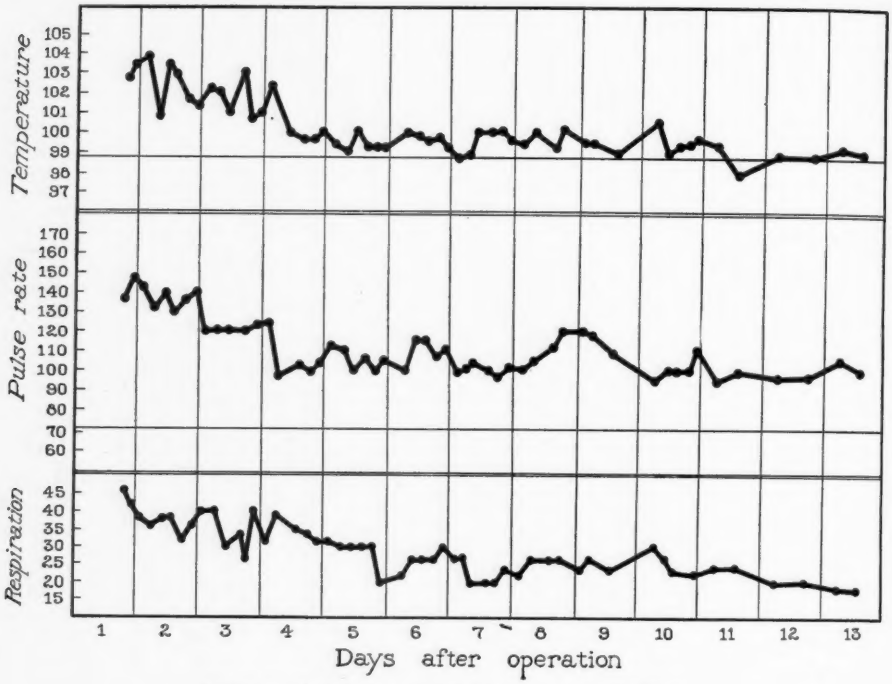
A white girl, 4 years 11 months of age, was admitted to the hospital November 28, 1934, complaining of abdominal pain. About two and one-half hours before admission, while playing, she had pulled a flower box, which measured about four feet by ten inches by ten inches (120 by 25 by 25 cm.), down upon herself. The box fell a distance of about three feet (90 cm.) with considerable force, pinning her beneath it. She was unable to extricate herself, but the box was removed after a few minutes. The child did not lose consciousness, and was able to arise and walk. Shortly thereafter she became nauseated and vomited. There was no hematemesis. She complained of some pain across the upper part of the abdomen, but this was not severe. An hour before she was brought to the hospital, the abdominal pain had become more severe, she had vomited again, had become cold and pale, and felt faint.

She presented a typical picture of shock; cold, moist extremities, marked pallor, respirations 46, thoracic in type. Pulse 136, weak and thready (Graph 1). The blood pressure could not be obtained (Graph 2). Immediate intravenous injection of 6 per cent solution of acacia was started, the foot of the bed elevated and heat applied to the extremities.

On physical examination no contusions, abrasions, or lacerations were found on the abdominal wall. There was tenderness, graded 3, throughout the abdomen, and rigidity, graded 3, of the abdominal muscles. The patient could not localize the abdominal pain to any particular area. Hepatic dulness was not obliterated nor was there any shifting dulness in the flanks. There was no abdominal distention, and the liver and spleen could not be palpated because of the tenderness and rigidity. The lungs were negative to percussion and auscultation. An occasional extrasystole could be heard over the apex of the heart. The reflexes were not abnormal and there was no paralysis. Erythrocytes 3,770,000, leukocytes 17,600, polymorphonuclears 55 per cent. According to the parents the patient had voided grossly normal urine before she came to the hospital. Urinalysis in the hospital disclosed albumin graded 1, a slight trace of sugar, and about six pus cells to the high power field. There were no erythrocytes in the urine. The child's condition

gradually improved, and after 350 cc. of solution of acacia had been given, her blood pressure was 110/60. Skin was warm. Pulse 136 but of better quality. It was apparent that she was suffering from a traumatic intra-abdominal lesion which required surgical treatment; therefore she was brought immediately to the operating room.

Under gas-oxygen-ether anesthesia a small right rectus incision was made, approximately five and one-half hours after the accident. As soon as the peritoneal cavity was opened, free fluid but no blood was encountered. Starting at the terminal part of the ileum, the entire small intestine was carefully examined, until about two inches (five cm.) distal to the ligament of Treitz, a perforation about one cm. in diameter was found. This was closed, and the area inverted with two continuous rows of chromic catgut. There was no evidence of injury to stomach, duodenum, large intestine, mesentery, liver, or spleen. It was felt safer to leave a small Penrose drain in the lower angle of the wound. The complete procedure took 35 minutes.



GRAPH 1.—Record of temperature, pulse and respiration.

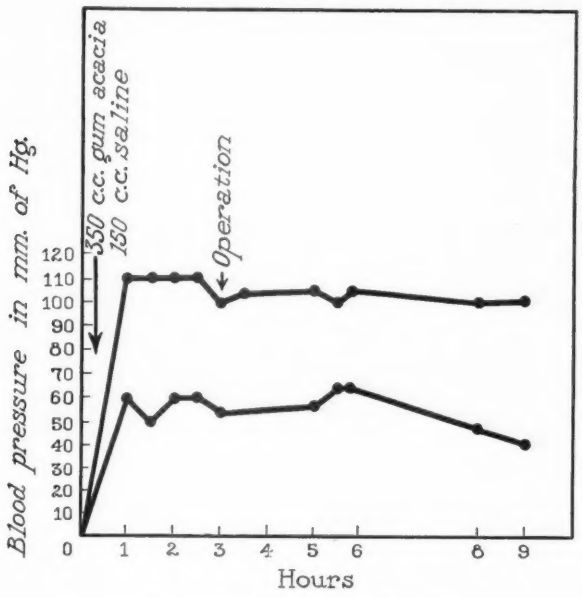
Fluids were given intravenously, and in increasing amounts by mouth, until, on the sixth day, the child was taking a liquid diet. The temperature and pulse rate gradually returned to normal. The drain was removed on the tenth day. Skin sutures on the twelfth and the silkworm tension sutures on the thirteenth days, when the patient was dismissed from the hospital.

No one sign is pathognomonic of intestinal rupture, but the diagnosis has to be made from the general clinical picture, including the history and a carefully performed physical examination. In most cases, pain is the outstanding symptom. It is present usually from the beginning, is constant in duration, dull, aching and not colicky in nature. It is diffuse throughout the abdomen early, and may then become localized. White[28] reported a case of ruptured jejunum, in which the pain was localized to an area below and

to the left of the umbilicus; the localization was similar to that of the pain of gastrojejunal ulcer.

A variable degree of shock is present. This may follow immediately after the injury, or may be delayed. Many reports of cases are found in the literature in which the patient felt well after the injury, except for the abdominal pain, and walked into the hospital apparently in good condition, only to develop the signs of shock later. With shock are present the cold, clammy extremities; rapid, thready pulse, and lowered blood pressure. Nausea and vomiting are present, and continued vomiting, especially after recovery from the initial shock, is a valuable early sign of intestinal perforation.

Tenderness is present but usually is of no aid in early recognition of



GRAPH 2.—Record of blood pressure.

the situation of the perforation, because it is usually diffuse. Rigidity of the abdominal muscles is probably the most valuable single sign of intestinal injury. It is present early in almost all the cases, is persistent, and even under the influence of morphine abates very little. Distention of the abdomen does not occur early, but is a manifestation of the peritonitis that later develops, and is a sign of poor prognosis when seen early.

Shifting dulness in the flanks early in the course is diagnostic of intra-abdominal hemorrhage, either from the mesenteric vessels or from the solid viscera. It is doubtful if, in early cases, enough intestinal content is present in the peritoneal cavity to produce this sign. Obliteration of the area of hepatic dulness, often mentioned by some as diagnostic of intestinal perforation, is seldom present in the early stages and is therefore useless as an early sign. One should look for it at the first examination, however, because its

detection at a later examination then becomes of diagnostic importance. Emphysema of the posterior abdominal wall is indicative of retroperitoneal rupture of either the duodenum or colon.

The pulse, temperature, blood pressure, and respirations are dependent on the presence or absence of shock. When the diagnosis is not obvious, a check on these vital signs every 15 minutes will often aid in establishing an early diagnosis, so that early surgical treatment can be carried out. The temperature, which at first is usually subnormal, rises after the patient recovers from the shock, and the pulse becomes slower and of better quality. A constantly rising pulse rate is of diagnostic importance, and if combined with an increased respiratory rate, surgical exploration is indicated.

Examination of the blood usually reveals moderate leukocytosis. When bleeding is present, either from the mesenteric vessels or from a solid viscus, the leukocytosis is more marked and there is a fall in the number of erythrocytes and in the hemoglobin content. Hourly examinations of blood should be made.

In regard to the diagnosis of intestinal perforation, then, it may be said that consideration of all the signs and symptoms is the important factor. No one sign or symptom is absolute, and no sign or symptom is to be disregarded. If a definite diagnosis cannot be made when the patient is first seen, careful observation, with repeated physical and blood examinations, as well as frequent determinations of the vital signs, should be carried out. It should be remembered that intestinal rupture may result from an apparently trivial injury, and also symptoms of rupture may be delayed. Cope,[5] in writing of the early diagnosis of this condition, stated that he would submit a patient to laparotomy if, in the absence of thoracic and renal injury, the following signs and symptoms were present: (1) severe abdominal pain persisting for six hours, accompanied by vomiting, gradually rising pulse rate, local rigidity, and deep local tenderness; or (2) absent or slight abdominal pain, with a steadily rising pulse rate, especially if the patient is restless and listless.

The importance of making an early diagnosis, and operating early, is seen from the figures presented by Siegel,[23] who studied the records of 376 cases in which operation was performed, and showed the increasing mortality as the time between the injury and the operation increases (TABLE III).

TABLE III

Mortality in Relation to Interval between Injury and Operation (Siegel)

Time of operation in relation to time of the injury	Mortality, per cent
Within the first 4 hours	15.2
From 5 to 8 hours	44.4
From 9 to 12 hours	63.6
After 12 hours	70.0

From TABLE IV it is seen that the total mortality in both cases operated and nonoperated is about 73 per cent, approximately the same as that for children less than 12 years of age, as is seen in TABLE II. The operative mortality derived from the totals is about 60.7 per cent (TABLE IV) and it is

TABLE IV

Data Concerning Subcutaneous Rupture of the Intestines of Patients of All Ages. The Quantities Include Those Given in Table II

Author	Cases	Patients operated on	Died	Operative mortality, per cent	Died without operation	Total mortality, per cent
Poland	61	0	0	00.0	61	100.0
Curtis	116	12	12	100.0	104	100.0
Siegel	376	376	194	51.6		51.0
Makins	21	15	12	80.0	6	85.7
Kirstein	18	18	10	55.5		55.5
Gage	85	40	23	57.5	45	80.0
Eisendrath[8]	40	40	21	52.5		52.5
Lund	19	15	8	53.3	4	63.1
Berry and Guiseppi	132	84	67	79.7	48	87.2
Tschistosserdorff	52	47	39	82.9	5	84.6
Lockwood	27	13	8	61.5	14	81.5
Battle	221	124	76	56.1	97	78.3
Stanley	4	4	1	25.0		25.0
Quain	12	12	5	41.7		41.7
Massie	34	24	17	70.8	10	70.6
Vance, 1923	12	5	5	100.0	7	100.0
Moynihan	61	47	35	74.4	14	80.3
Vance, 1928	10	10	5	50.0		50.0
Cooke	12	1	1	100.0	11	100.0
Totals	1,313	887	539	60.7	426	73.4

about the same, 58.8 per cent, for children (TABLE II). The high mortality is accounted for chiefly by the late operations, when peritonitis is developing or has developed. However, another reason for the high operative mortality is that patients are in a state of severe shock. Preparation of patients who are in shock is an important part of the treatment, and one of us (Counseller[6]) previously has pointed out the efficacy of 6 per cent solution of acacia in treating shock immediately, whether or not hemorrhage has taken place. Martin[16] has pointed out that following penetrating wounds of the abdomen, the important complications, in order, are shock, peritonitis, and hemorrhage. This is also true following subcutaneous injuries of the abdomen. Fraser and Drummond[9] have expressed the belief that very little intestinal leakage occurs from the perforation during the first six hours because peristalsis is absent and because the everted mucous membrane acts as a plug. Even in complete tears, according to the belief they expressed, the leakage is not great during the early hours, because of the contraction of the circular muscle layer in the severed ends. This substantiates the belief that immedi-

ate operation on a patient who is in shock only adds insult to injury, and that it is better to improve the patient's condition first by means of measures to combat shock. The immediate treatment then becomes the treatment of shock when it is present, but if shock is absent, one should not wait for it to appear but immediately perform laparotomy.

The outlook is better for patients whose perforations are high in the intestinal tract than it is if the perforations are lower, because of the relatively cleaner intestinal content of the higher segments, and hence the less likelihood of peritonitis developing. However, high tears are less likely to cause such early and striking symptoms as those in the lower part of the tract, so patients whose tears are high usually come to operation later. Among children who have sustained visceral injury, the younger the child, the more serious the outlook.

In carrying out the surgical treatment, that procedure which can be done most quickly, with the least amount of shock to the patient and trauma to the tissues, should be chosen. The operation should not be prolonged, and a constant check should be kept on the blood pressure, pulse, and respiration of the patient, so that immediate treatment can be undertaken if shock supervenes. Long time-consuming resections should be avoided unless the viability of the injured intestine is questionable.

The abdomen may be opened by an incision through either rectus muscle, to give the best exposure, unless localizing signs are present which indicate the section of the bowel involved. In these cases, the incisions may be placed to facilitate repair.

After the peritoneum has been opened, if active bleeding is present, it is better to bring the intestines outside the abdomen and immediately to control the bleeding. If there is no hemorrhage, it is better to examine the bowel and mesentery minutely, starting at some fixed point such as the ileocecal region, and drawing several inches of bowel out at one time, following up or down the whole intestinal tract. When a perforation of the bowel is found it is better to cover it with a warm, moist sponge and lay it aside until the whole intestine has been examined. Sometimes, resection of a portion of bowel may be necessary because of multiple lesions close together, or because of a damaged blood supply in the mesentery, and if a perforation has been closed and then it is found that resection of the loop containing the closed perforation is necessary, much valuable time has been lost.

The question of drainage is an individual one, and depends on the conditions found at operation. The same principles apply in these cases as in all operations on the abdomen.

The postoperative care is concerned with seeing that the patient is kept comfortable by the use of sedatives, and that the intake of fluid is adequate; fluid may be introduced by the intravenous, subcutaneous or rectal routes. After operation, feeding by mouth may be started with sips of water and the amount and consistency of the ingesta may be increased as the patient

tolerates the increase; gradual progress is made through liquid and soft to a normal diet.

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THE INDICATIONS FOR GASTROSTOMY AND JEJUNOSTOMY

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It is generally agreed that the Janeway, or any good gastrostomy, works admirably in the presence of obstructive growths of the pharynx and of many of those of the esophagus, after stricture of the esophagus, and following pressure upon the gullet from mediastinal tumor. There are, however, certain destructive growths of the esophagus, particularly those of the distal portion, and of the cardia after which a gastrostomy continues to leak (Fig. 1).

It is recognized that a gastrostomy in a carcinomatous stomach facilitates the radiologist in implanting radium in the gastric tumor. This, too, may be accomplished through a gastrotomy following which a jejunostomy may be performed and the stomach allowed to remain at rest. If, however, some special indications call for repeated approach to a gastric growth through a gastrostomy, one must occasionally choose between its advantages and the disadvantages of gastric incontinence.

The combination of gastrostomy and pathology in the cardiac end of the stomach involving the lesser curvature has repeatedly demonstrated incontinence of the gastrostomy. There appear to be two essential factors in this lack of control of the fistula; namely, the impairment of the gastric vagomotor nerves and the resultant digestive effects upon the abdominal wall about the fistula. The loss of fluids and skin irritation so aggravate the distress of the patient that the surgeon may feel compelled to attempt closure of the gastrostomy.

Encroachment upon the gastric vagus, particularly fibers entering into the pyloric branch, may either stimulate these fibers so as to produce a vagotonic stomach or destroy these fibers so as to divorce the pyloric portion from the fundic part of the stomach, thus producing a diffuse pylorospastic stomach. The former condition is depicted in the psychopathic patient with vagotonia[1, 2, 3] (Fig. 2), with eructations and vomiting as prominent symptoms. The latter condition is represented by the stomach after annular resection.[4] In both of these nervously impaired stomachs it is believed that the intragastric pressure is markedly increased. The fistula presents a means of least resistance and a site for regurgitant fluids. Occasionally, after gastrostomy feeding, such a patient experiences epigastric pain synchronous with painful gastric contractions (at 15 to 20 second intervals) and immediately following these spasms one may observe jets of gastric contents from the mouth of the fistula. These patients cannot vomit any more than they can swallow but they do pass through the early painful stage of the act of vomiting.

Radiographs of these individuals indicate spasms of the pylorus or stomach characteristic of vagotonia. Some of the chyme passes on into the duo-

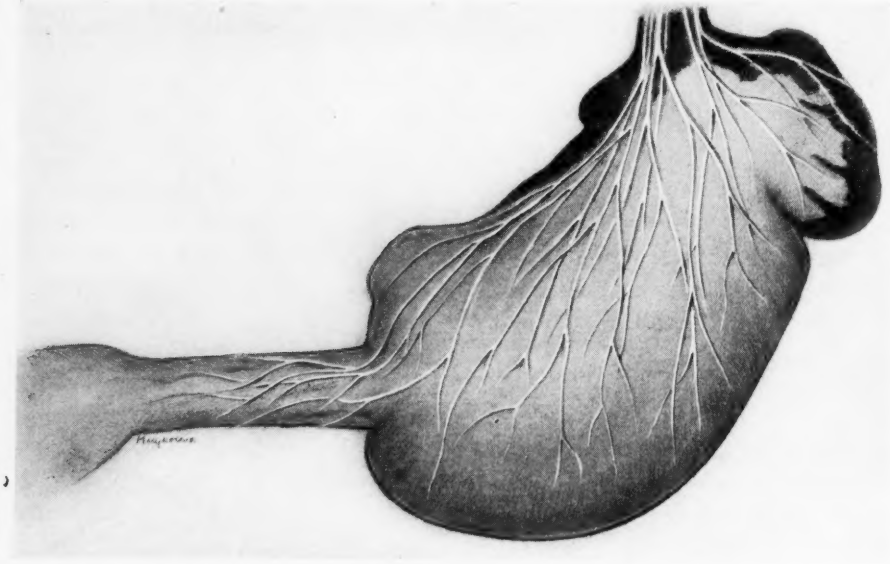

FIG. 1.—Drawing of recent case representing malignant growth at cardia and lesser curvature. The left vagus is sketched in to indicate relation of nerve fibers to tumor. Gastrostomy in this patient was followed by gastric colic and incontinence at stoma.

denum and when once beyond the pyloric sphincter continues on through the intestine. Large doses of belladonna, in certain cases at least, are of no avail in lessening the gastric colic. Possibly the impairment or destruction of the

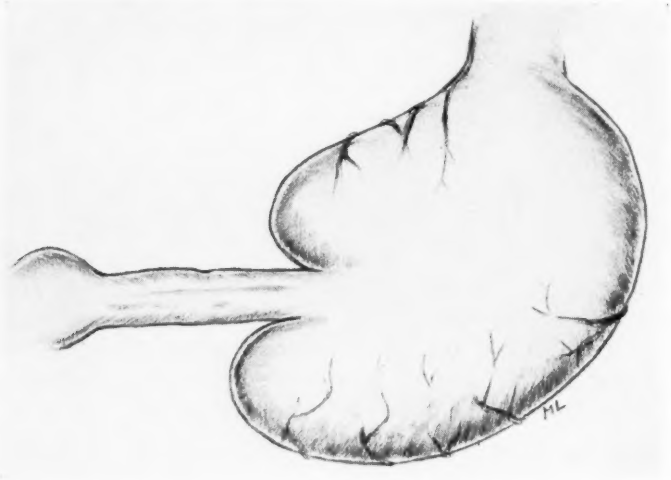

FIG. 2.—Drawing of vagotonic stomach in psychopathic patient with marked regurgitation and vomiting. Diffuse pylorospasm was observed on radiogram and in open abdomen.

vagus nerve at the cardia renders ineffective the action of atropine upon the termination of this nerve within the stomach wall. These unfavorable

stomachs occur infrequently and are thought to be vagotonic from mediastinal or gastric irritation.

Wolfer[5] has published a similar conclusion regarding the wisdom of performing jejunostomy rather than gastrostomy for these lower esophageal cancers, chiefly because of the severe colicky pyloric pain experienced in such cases.

This condition of gastric malfunction is not to be mistaken for the too frequent instances in which acute dilation follows operation. In this latter condition the operative procedure may have been carried out smoothly and expeditiously and the patient returned to bed in a very satisfactory state. Proper feedings in proper amounts may have been regularly administered; but if one catheterizes the gastrostomy he may obtain as much as two quarts of foul material. It is remarkable because the stomach of such an individual at laparotomy is often small, atrophic, and apparently incapable of such dilation. This can be prevented or relieved by instructing the nurse in charge to aspirate or to gently irrigate the stomach before the introduction of each feeding.

The principle of rest for a diseased stomach would seem to contra-indicate using such a stomach as a feeding reservoir. It is the practice on the Third Division, Bellevue Hospital, after determining inoperable malignant involvement of the stomach, either to close the abdomen at once or to perform jejunostomy.

In those instances of destructive esophageal growths in which it is feared that motor gastric disturbances will interfere with satisfactory emptying at the pylorus after gastrostomy, one may add section of the branches of the principle anterior nerve on the stomach wall.[6] This has been carried out in several such cases with apparent encouraging results.

If one desires to explore manually it is thought wise to make a median or right paramedian laparotomy and use the left rectus by means of a stab wound as the site for the jejunostomy. The exploratory wound may then be closed independently and protected by the dressings from exposure to digestive juices. This is important because if jejunal secretions seep into the depths of the wound necrosis and protein absorption follow with high temperatures, rapid pulse, pallor, *etc.*, suggestive of the "shock" syndrome following protein intoxication. Preserving the rectus fibers provides some sphincteric control and guards against wound contamination. In most instances observations before operation suffice to disclose the degree of metastasis so that the incision for jejunostomy is all that is necessary.

From an examination of the case records of the Third Division at Bellevue Hospital the past five years (1930–1934), there appear 43 cases of gastrostomy. Of these ten succumbed so soon following operation as to make a determination of the functioning of the gastrostomy impossible. Of these, five died of shock, four of peritonitis and one of pneumonia.

It is interesting to note that these cases have been increasing in number, there being none in 1930, seven in 1931, 22 in 1932–1933 and 14 in 1934.

Of the 33 cases studied, five were considered not working well from the standpoint of gastrostomy, and 28 as functioning satisfactorily. An examination of the five unsatisfactory cases indicates that the lesion was at the cardia in three (60 per cent) and in the esophagus above the cardia in two (40 per cent). In the 28 successful gastrostomies, in five (18 per cent) the pathology was at the cardia, four (14 per cent) in the lower third of the esophagus, seven (25 per cent) in the middle third, four (14 per cent) in the upper third, and eight (28 per cent) in the pharynx (Figs. 3 and 4).

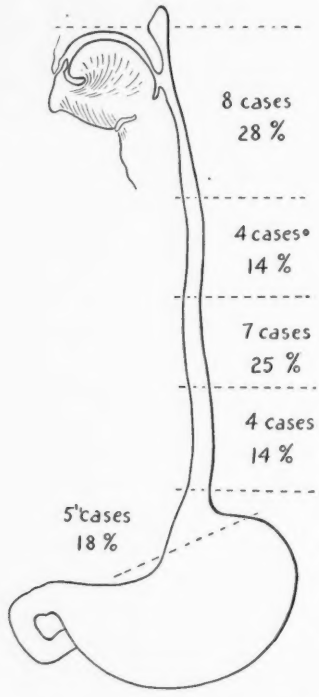

FIG. 3.—Diagram based on Table I representing location of esophageal disease for which gastrostomy was performed and worked well. Note 18 per cent located at cardia and 82 per cent above.

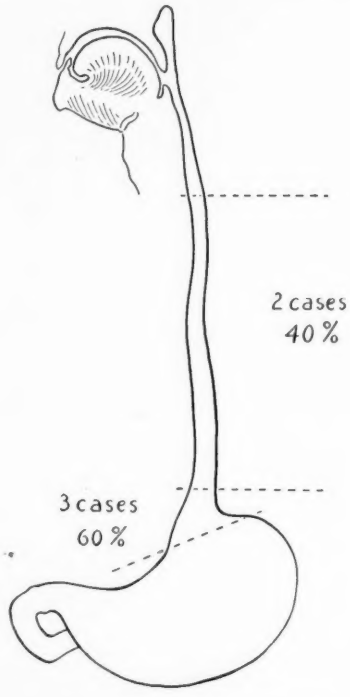

FIG. 4.—Diagram representing distribution of malignant growths for which gastrostomy was performed and worked unsatisfactorily. Note 60 per cent at cardia and 40 per cent above.

There were 13 jejunostomies from 1932 to 1934 for inoperable gastric cancer. Of these, six were of the type described by the author in 1932.[7] This technic embraces jejuno jejunostomy and a two-barreled jejunostomy. The *temporary* jejunostomy has been found to work well in the acute cases or cases requiring jejunostomy for a short period of time. In these, the muscle-peritoneal flap combined with double inversion about an indwelling catheter and omental grafting has been found advantageous to the more common older methods. The *permanent* method lessens the incidence of postoperative mortality, does away with the fear of having the catheter come out and of the danger of replacing it, and by presenting two openings for

TABLE I

*Correlation of Gastrostomy Function and Location of Pathology**

		Location	Operation	Function	Disposition
		1931			
1.	J. T.	Carcinoma Esophagus Stomach (lesser curvature)	Janeway (L.)	Works well	Discharged 48 days postoperative
2.	R. S.	Carcinoma Esophagus (upper ⅓)	Janeway (mod. W.)	Works well	Discharged 30 days postoperative
3.	B. H.	Carcinoma Esophagus (upper ⅓) O-T fistula	Janeway (B.)	Worked well	Died 12 days postoperative. (Hemoptysis)
4.	A. S.	Carcinoma Esophagus (mid. ⅓)	Janeway (L.)	Works well	Discharged 31 days postoperative
5.	H. M.	Carcinoma Esophagus Stomach	Janeway (mod. N.)	Works well	Discharged 31 days postoperative
6.	J. M.	Carcinoma Esophagus Stomach	Janeway (L.)	Works well	Discharged 18 days postoperative
7.	W. S.	Carcinoma Esophagus (lower ⅓) Stomach	Janeway (L.)	Worked well	Died 17 days postoperative (pneumonia)
		1932–1933			
8.	C. J.	Carcinoma Esophagus (mid. ⅓)	Janeway (B.)	Acute dilation Stomach Regurgitation	Discharged 14 days postoperative
9.	W. M.	Carcinoma Esophagus (upper ⅓)	Janeway (N.)	Works well	Discharged 13 days postoperative
10.	J. L.	Carcinoma Larynx Tracheotomy	Janeway (B.)	Works well	Discharged 13 days postoperative
11.	C. G.	Carcinoma Nasopharynx Tracheotomy	Janeway (B.)	Works well	Tracheotomy closed. Discharged 72 days postoperative for continued radiotherapy
12.	T. C.	Carcinoma Pharynx (rt. tonsil)	Janeway (B.)	Works well	Discharged 17 days postoperative
13.	B. H.	Carcinoma Esophagus	Janeway (mod. C.)	Works well	Discharged 2 months postoperative. Radiotherapy
14.	M. P.	Carcinoma Stomach Carcinomatosis	Janeway (mod. S.)	Regurgitation	Died 17 days postoperative (perforative gastritis)

TABLE I (*Continued*)

		Location	Operation	Function	Disposition
15.	V. G.	Carcinoma Esophagus (mid. ⅓) O-T fistula	Janeway (mod. L.)	Worked well	Died 20 days postoperative. Lung abscess. Subdiaphragmatic abscess. Peritonitis
16.	H. M'C.	Stricture Esophagus	Janeway (B.)	Works well	Seven months' treatment of stricture; then closure of gastrostomy
17.	G. S.	Carcinoma Esophagus (mid. ⅓)	Janeway (L.)	Worked well (regurgitation)	Died 24 days postoperative (pneumonia)
18.	H. S.	Carcinoma Esophagus (mid. ⅓) O-B fistula	Janeway (mod. C.)	Worked well	Died 25 days postoperative (pneumonia)
19.	J. R.	Carcinoma Esophagus (lower ⅓)	Janeway (mod. C.)	Worked well	Died 11 days postoperative (cachexia)
20.	J. B.	Carcinoma Esophagus (lower ⅓)	Janeway (mod. N.)	Works well	Discharged 30 days postoperative
21.	E. K.	Stricture Esophagus	Janeway (B.)	Works well	Four months' treatment stricture, then gastrostomy closed
22.	M. R.	Carcinoma Esophagus	Janeway (mod. L.)	Works well	Discharged 57 days postoperative
23.	P. R.	Carcinoma Esophagus (lower 4 cm.)	Janeway (B.)	Works well	Discharged 37 days postoperative
			1934		
24.	H. W.	Carcinoma Stomach (lesser curvature)	Janeway (mod. L.)	Pylorospasm. Regurgitation	Discharged 2 months postoperative
25.	P. R.	Carcinoma Stomach (cardia)	Janeway (B.)	Regurgitation	Discharged 13 days postoperative
26.	I. H.	Carcinoma Esophagus	Janeway (B.)	Regurgitation	Died 46 days postoperative
27.	E. S.	Carcinoma Esophagus Stomach	Janeway (B.) Vag. Sect.	Worked well	Died 30 days postoperative
28.	C. W.	Squamous Cell Epithelioma Nasopharynx	Janeway (B.) Vag. Sect.	Works well	Discharged 11 days postoperative
29.	F. P.	Carcinoma Esophagus (mid. ⅓)	Janeway (B.) Vag. Sect.	Works well	Discharged 13 days postoperative
30.	J. C.	Carcinoma Larynx Esophagus	Janeway (B.) Vag. Sect.	Works well	Discharged 18 days postoperative
31.	W. L.	Carcinoma Esophagus (mid. ⅓)	Janeway (mod. L.)	Worked well	Died 10 days postoperative (pneumonia)

TABLE I (*Continued*)

		Location	Operation	Function	Disposition
32.	E. M.	Carcinoma Esophagus (lower ¼)	Janeway (mod. C.)	Works well	Discharged 19 days postoperative
33.	J. F.	Carcinoma Tonsil Larynx	Janeway (mod. C.)	Worked well	Died 16 days postoperative

* This table includes two cases of stricture of the esophagus, *Cases* 16 and 21, and 31 cases of malignancy.

jejunal feeding instead of one, divides up on the traumata of tubal feedings. Jejuno jejunostomy combined with jejunostomy provides for the entrance of digestive secretions from the stomach and duodenum into the jejunum below the stoma and does away with the possibility of technical intestinal obstruction. It is generally understood that these cases being of advanced malignancy have practically no life expectancy so that the object of any palliative procedure can be to prolong life but a few days and make these few days more endurable. At the present writing it is believed the Janeway gastrostomy in the pharyngeal and esophageal cases and the permanent jejunostomy for the esophago gastric and certain of the inoperable gastric growths best ensure the desired results.

These operations have been performed by many different surgeons on the Third Division so that the results more typically represent those of general surgical practice in a city hospital. It will be observed that, during 1934, four gastric vagotomies were performed in conjunction with the Janeway gastrostomy. The effect upon gastric function favorably impressed those of us who observed these cases daily, but no conclusions are to be drawn on these few observations at this time.

CONCLUSIONS

(1) Gastrostomy (Janeway) is recommended for carcinoma and obstruction of the pharynx and esophagus.

(2) Gastric vagotomy may be added to gastrostomy for a destructive lesion of the lower half of the thoracic esophagus.

(3) Jejunostomy has been used in this series in inoperable gastric carcinoma not considered entirely hopeless.

(4) Both temporary and permanent jejunostomy has its indications in the inoperable or not primarily resectible ulcers and new growths of the stomach. The technics already published give best results at the present time.

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TORSION AND VOLVULUS OF THE STOMACH WITH DIAPHRAGMATIC HERNIA*

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TORSION and volvulus of the stomach are of engaging interest because of the rarity of the total variety, there being only about 70 cases reported in the literature, the obscurity of the etiology of both total and partial and a growing interest in the diagnosis of the partial and intermittent group.

Great emphasis has been placed by most writers on terminology and classification. Becher states that true volvulus means rotation of the whole viscus between pylorus and cardia, while torsion takes place around the other axis. Von Haberer suggests volvulus mesenterio-axialis and volvulus organo-axialis. All recognize total and partial varieties. In describing them they use interchangeably the terms twisting and rotation. Torsion is derived from the Latin *torqueo* meaning to twist. Volvulus comes from the Latin *volvo* meaning to roll. It seems inconsequential from either a descriptive or etiologic basis whether the stomach twists or rolls itself. Obviously in either case rotation has occurred. It would seem, then, for the sake of accurate description, clarity and simplicity, the term rotation of the stomach be used. We may then describe partial or complete rotation which may be around either the long or transverse axes of the stomach, from right to left or left to right in rotation around the transverse axis, or anterior or posterior in those cases of rotation around the long axis and those that rotate around both axes (Figs. 1, 2 and 3).

Many explanations have been offered as to etiology, but the exact mechanism by which rotation occurs is still obscure. The conditions in which either partial or complete rotation has occurred are well described. In all the cases there has been a lengthening of the mesenteries, particularly the gastrohepatic. This lengthening may be the result of a general ptosis. In congenital abnormalities as in the case reported by Gorell in which there was complete rotation around the traverse axis, the great omentum was only an inch long and did not contain the transverse colon, the transverse colon having a mesentery of its own behind the stomach, the gastrohepatic omentum was five inches long. Diaphragmatic hernia is undoubtedly a predisposing cause as the stomach is drawn upward resulting in lengthening of the mesenteries.

Payer reported 500 cases of diaphragmatic hernia in which there were 12 cases of rotation of the stomach. Bonafé and Poulain report two cases of rotation of the stomach following phrenicectomy, the same mechanism as in

* Read before the Philadelphia Academy of Surgery, November 5, 1934.

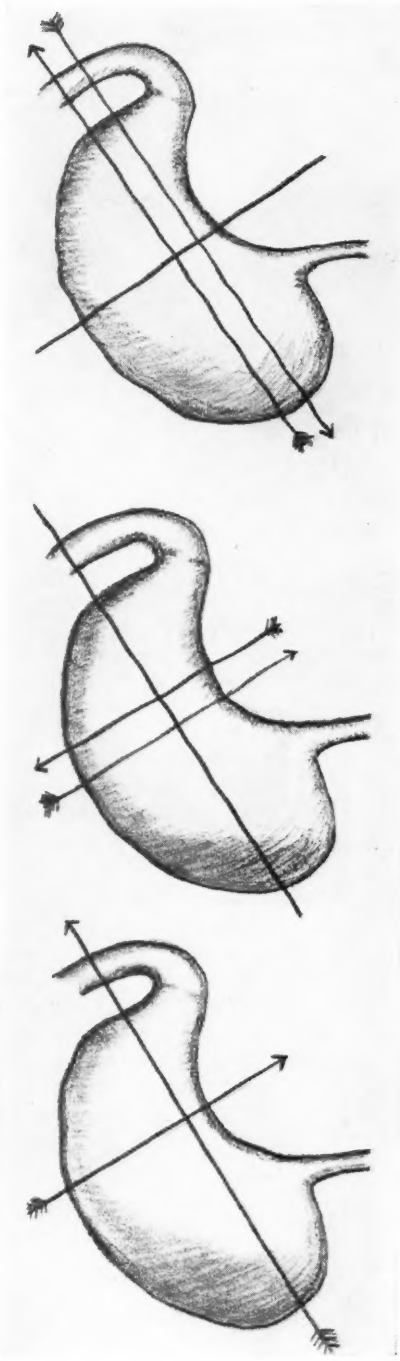

FIG. 1.—Rotation around the transverse axis. (After Kocher.)

FIG. 2.—Rotation around the long axis. (After Kocher.)

FIG. 3.—Rotation around the transverse and long axes as it occurred in this case.

diaphragmatic hernia in all probability operating to lengthen the mesenteries following this procedure.

In the cases of partial rotation some gastric condition, particularly hourglass stomach, gastric ulcer or gastritis, seems to be present usually. The exciting causes suggested have been overfilling of the stomach, gaseous distention, vomiting, hyperperistaltic waves, weakness of the abdominal wall and trauma.

Diagnosis of partial rotation has been made in several reported cases because of its characteristic symptoms and signs and the roentgenologic appearance. Total rotation should be suggested when there is severe acute abdominal pain with vomiting which suddenly stops followed by retching and immediate abdominal distention and shock. There is inability to swallow and to pass a stomach tube. The diagnosis may be confused with that of a ruptured viscus, intestinal obstruction, acute pancreatitis, mesenteric thrombosis, and coronary occlusion.

Immediate operation is urgently indicated, reducing the rotation and performing a gastropexy. The mortality in complete rotation is about 50 per cent, the percentage being higher in the rotations around the transverse axis.

H. W., married, white male, aged 65, was admitted to the Germantown Hospital on Doctor Swartley's service, July 25, 1934, with the chief complaint of severe pain in the abdomen. He had been perfectly well until 6 P.M. when he ate freely of corn fritters. Acute generalized abdominal pain followed within a few minutes and was very severe. He vomited and then took magnesium sulphate, which he also vomited. After that he could not vomit but retched frequently. The past history revealed that he had had several attacks of "indigestion," the most severe one being last Thanksgiving Day. The patient's appearance was that of profound shock—great pallor with cyanosis and rapid labored breathing. His temperature was subnormal, pulse 150 and respirations 48. The blood pressure was 30/0. The breath sounds over the left chest anteriorly and posteriorly were shallow and occasional râles were heard over the base. The percussion note was flat over the left base posteriorly extending to the fifth rib. The heart sounds were distant but the apex beat did not seem to be much displaced. The abdomen was moderately distended. There was tenderness over the epigastrium and some in the lower right quadrant. Marked increased resistance was found throughout the abdomen, particularly in the epigastrium. No masses were palpable. Peristalsis was absent throughout the abdomen. Hemoglobin 80 per cent; red blood cells, 5,030,000; white blood cells, 17,600; neutrophiles, 80 per cent; large lymphocytes, 19 per cent; mononuclears, 1. A specimen of urine was not obtained.

The tentative diagnosis was perforated gastric ulcer; that of coronary occlusion was temporarily entertained. Immediate operation was decided upon, but the patient died before it could be instituted, death occurring six hours after the onset of pain.

Autopsy report by Dr. Frank Lynch. "The left pleural cavity is partly filled with bowel which has herniated through thinned out diaphragm, the left lung occupying about two-fifths of its normal space. The heart is pushed slightly to the right and appears to be slightly smaller than normal. Coronaries are slightly tortuous but soft. The stomach lies in the middle of the abdomen and dilated to such an extent that it reaches almost to the pubic arch. The posterior wall presents anteriorly, the pyloric end to the left and the fundus to the right, with the greater curvature higher than the level of the lesser. By rotating the stomach from above downward and from right to left it falls back into its normal position. It was purplish-brown in color and partly

filled with liquid material. While the stomach was in the rotated position, the spleen was visible and slightly to the right of the midline. The major portion of the small intestines were above the stomach and some contained within the hernial sac. The cecum was drawn upward toward the liver and a large part of the transverse colon with the omentum were found in the hernial sac. On opening the stomach it was found to be filled with debris, mucus, fluid and a slight admixture of blood. On emptying the stomach, it was found to be thickened, hemorrhagic and covered with a large quantity of thick mucus. The pancreas is widened at the proximal end, the tail is narrow and flabby.

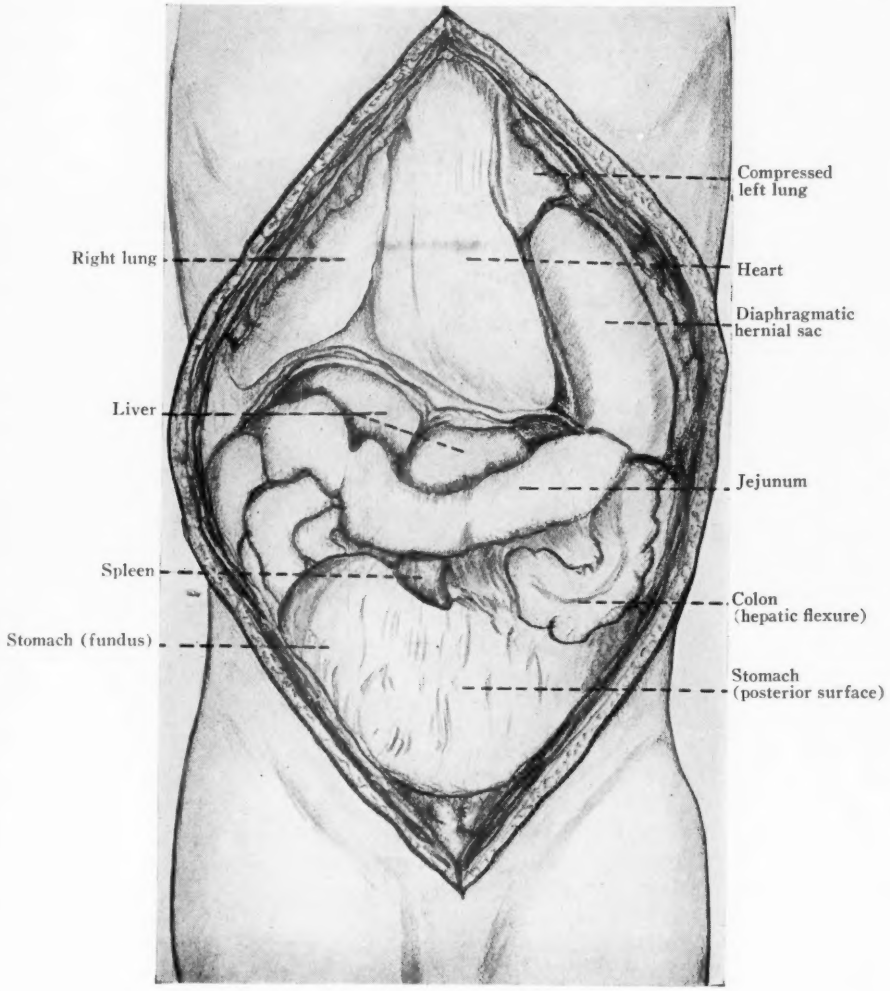

FIG. 4.—Showing the abnormal arrangement of the viscera.

Congestion of the proximal end is due probably to venous stasis. The gastrohepatic omentum is elongated and the duodenum is completely invested with peritoneum forming a mesentery (Fig. 4)."

It will be seen from this description that in all probability the diaphragmatic hernia was the cause of the elongation of the gastrohepatic omentum as the stomach conveniently occupied and adapted itself to the hernial sac when

the intestines and omentum were removed and the rotation of the stomach relieved. Whether or not the duodenal mesentery was formed by traction or was a congenital abnormality cannot be stated. The rotation of the stomach was a complete rotation around both axes, around the longitudinal axis anteriorly and around the transverse axis from left to right. It is interesting to speculate on the exciting cause of the rotation. The fact that the omentum was found well in the hernial sac suggests the possibility that it may have initiated the rotation of the stomach by migrating into the hernial sac, drawing on the gastrocolic omentum which in turn drew the greater curvature of the stomach upward bringing about, together with unusually active peristalsis from overloading the stomach with indigestible food, the resultant rotations.

INVISCERATION*

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CASE REPORT.—F. K., Male, aged 56, was admitted to the Medical Division of Mt. Sinai Hospital, October 31, 1933, with the chief complaint of sense of pressure and of pain in the epigastrium radiating to the right scapula. The onset was about two months earlier, with mild symptoms which became more severe. The pain began after the morning bowel movement and lasted one or two hours, then recurred after the evening meal. It was not very regular nor consistent. Occasionally it was accompanied by vomiting. Relief was obtained by the use of a hotwater bag and by certain powders. There had been a loss of about 30 pounds in a year. The patient had had diarrhea for the last seven years. Appetite was fair; there was occasional pyrosis and belching, but no particular food intolerance. He was treated in the Gastro-Enterological Out Patient Department five years ago, and one year ago, with good results. History was not otherwise significant.

Examination.—The patient was a thin, undernourished man, garrulous and hypochondriacal. General examination showed no abnormalities of any relevance save for an abdomen of ptotic type with a point of tenderness in the right hypochondrium near the midline. Blood count and chemistry, Wassermann, urine and icterus index were within normal limits. Orthodiagram revealed the heart to be normal in position and rather small in size. Gastro-intestinal radiographic study showed a penetrating ulcer of the lesser curvature. Outside of a generally low position of the stomach and colon, the abdominal viscera were visualized as substantially normal.

A diagnosis of gastric ulcer was made and the patient transferred to the Surgical Service of Doctor Eliason, who operated November 16, 1933, through an upper right rectus incision. The peritoneal cavity was entered without difficulty and was found to be *entirely empty*. The cavity was lined with an unbroken membrane having the appearance of ordinary smooth peritoneum. It began above at the transverse fissure of the liver and as the lesser omentum extended to the lesser curvature of the stomach. The stomach was covered by this continuous sheet which was firmly adherent to its anterior surface. From the greater curvature of the stomach the peritoneum extended in an unbroken sheet down to the promontory of the sacrum, whence it passed anteriorly in the horizontal plane to the anterior abdominal wall and became continuous with the anterior parietal peritoneum. Thus it was impossible to introduce the exploring hand into the true pelvis. This peritoneal sheet extended unbroken to both lateral abdominal walls and was continuous with their parietal peritoneum. Beneath it could be seen the great omentum overlying all the intestine except a few coils of small bowel in the left lower quadrant. Nowhere did any portion of the intestine, large or small, project through it into its cavity. It was not determined definitely whether any of the subjacent bowel was adherent to it, but its smooth unwrinkled appearance made this appear improbable.

The stomach was carefully examined and no evidence of ulcer found. The abdomen was then closed without further investigation. The patient made an uncomplicated convalescence, and when seen in the follow up clinic stated that he had only occasional abdominal discomfort, but that his tendency to diarrhea continued.

In order that the various theories entertained as to the nature and causation of the condition described be clear, a brief summary of the develop-

* Read before the Philadelphia Academy of Surgery, November 5, 1934.

ment of the peritoneum may be given. The peritoneum develops from the mesenchyme. The portion of it covering the abdominal wall requires no description. The mesenteries and visceral peritoneum are formed from the anterior and posterior primary mesenteries. The former extends in the sagittal plane from the anterior surface of the upper portion of the gut-tube to the anterior wall only as far down as the umbilicus, and carries in its free edge the umbilical vein. In adult life it forms the peritoneal covering of the liver and its falciform ligament, as well as the lesser or gastrohepatic omentum. With the rotation of the stomach the anterior mesentery changes its axis from the vertical to the horizontal and is drawn to the right.

The posterior mesentery, usually called the mesogastrium, extends in early fetal life from the posterior surface of the gut-tube to the anterior surface of the aorta. It is this mesogastrium which later forms the great omentum, transverse mesocolon, mesosigmoid, mesentery of the small intestine and the visceral peritoneum. Its development and relations, including as they do the formation of the lesser peritoneal cavity and the numerous peritoneal fossae, are too complex to be described in this short presentation. It may be mentioned, however, that the great omentum is in fetal life, and sometimes at birth, an open sac communicating with the lesser peritoneal cavity, and that at this time it depends directly from the greater curvature of the stomach. At about the time of birth or shortly thereafter, its anterior and posterior leaves fuse with each other. This results in the obliteration of the so called omental bursa, the formation of the gastrocolic omentum, and the attachment of the great omentum to the transverse colon.

Various possible explanations of the situation found in our patient are to be considered. Diaphragmatic hernia may be discarded at once for the obvious reason that there was no evidence of its existence. A more plausible theory is that of intraperitoneal hernia. This is a condition of no great rarity, in which much or indeed most of the intestine finds its way into one of the numerous peritoneal fossae and comes to be retroperitoneal in appearance at least. When the abdomen is opened the cavity may seem to be empty, but closer investigation will always show at least a portion of intestine, either small, large or both, to lie free within it. This was not true in the present instance and therefore hernia into a peritoneal fossa is excluded.

Cases have been recorded in which there was double anterior peritoneum. This, however, has always been of limited extent, usually in either the right or left lower quadrant, and a potential cavity only in that the two layers lay apposed. Furthermore, when the deeper of these layers has been penetrated, the ordinary intra-abdominal relationships obtain. It has been suggested, likewise, that the true peritoneal cavity may not have been entered in our case, but that only the cleavage line between the transversalis fascia and the peritoneum may have been reached. This hypothesis is effectually excluded by reason of the facts that the free edge of the liver could be seen and that the hand could be passed down to the sacrum, though not beyond it.

Another theory, based upon a mechanism analogous to that by which the second and third portions of the duodenum become retroperitoneal, has

been advanced. For various reasons it may be discarded. Among these is that it is incompatible with the existence of a great omentum and that the abdominal viscera not completely covered by peritoneum are adherent to the posterior abdominal wall. It is hardly conceivable that the entire intestinal tract should be so adherent.

A final concept is that we are dealing with an extraordinary omental bursa, that is, a persistence of the fetal condition of nonfusion of the anterior and posterior layers of the great omentum. In addition, we must postulate, in order to fulfill the conditions found at operation: (*a*) a fusion of the anterior layer of the great omentum with the anterior parietal peritoneum at a point a short distance below the greater curvature of the stomach; (*b*) a loss of the fatty and vascular character of this layer; (*c*) an attachment of the omentum to the lateral abdominal walls so as to form a complete septum and to the promontory of the sacrum below; (*d*) an assumption by the anterior surface of the posterior layer of the omentum of the characteristics of ordinary peritoneum and the disposal of the fat and blood vessels upon the posterior layer; (*e*) that the incision traversed the anterior layer of the omentum as the latter passed from the greater curvature to the abdominal wall.

Of these postulates, that concerned with the existence of an omental bursa and that which provides for the attachment of the omentum to the lateral abdominal walls are not without precedent. As regards the remaining ones, no precedent has been found for the fusion with the anterior wall; the suggested alteration in the arrangement of the omental fat seems to be inconsistent with the known anatomy of the omentum; and finally, the incision was not observed to traverse an anomalous layer of peritoneum between the greater curvature and the anterior parietes.

It is now apparent that no tenable explanation for the findings in this case has been offered. Not only is this true, but upon the accepted facts of the embryology and anatomy of the peritoneum we have been unable to construct one. A search of the literature has not yielded either an identical or a comparable case. Whether our failure to provide an answer to our riddle is due to faulty or incomplete observation, or to a hitherto undescribed anomaly is of course an open question. Pending enlightenment we have tentatively suggested the name "Invisceration" and are reporting the case in the hope that it may provoke discussion which will dispel what to us is an unsolved mystery.

Discussion.—Dr. Benjamin Lipshutz (Philadelphia) said that a sound and tenable explanation of this extremely rare peritoneal anomaly presented by Doctor Goldsmith, under the title "Invisceration," is best obtained by a consideration of certain well established embryologic conditions. For there is nothing here to indicate that the anomaly might have been the result of a pathologic condition. All of the special features suggest an early embryologic origin of this anomaly. In order to accurately interpret such an anomaly it is necessary to make careful observations of the various peritoneal attachments, to note displacements of viscera, and the related blood supply. Such observations are possible only at autopsy or in the dissecting

room. Variations in the rotations of the bowel are well known—an instance of non-rotation or faulty rotation was reported by Doctor Klopp, before this society some years ago; peritoneal invisceration, on the other hand, is an entirely different anomaly. The explanation which would seem to best clarify the reported case of invisceration is that advanced by James W. Papez of Cornell.

This anomaly is interpreted as one of early embryonic origin, by assuming that the abnormal peritoneal sac was derived from the normal embryonic umbilical hernia, which encloses the small intestines prior to the 30 Mm. stage of the embryonic growth. The bowel in the embryonic normal umbilical hernia grows and develops and produces bulbous enlargement of the proximal end of the cord. The umbilical hernia is lined with peritoneum which is continuous with the peritoneum of the abdominal cavity. It is believed that the neck of the sac at the umbilical orifice becomes prematurely adherent to the duodenum by virtue of close opposition, which exists between them at this early stage of development.

The retracting forces which suddenly draw the intestines into the abdominal cavity also draw in the peritoneum of the early embryonic cavity, in case an adhesion happens at the umbilical orifice. Within the abdominal cavity, the small intestine grows, expanding the sac to its adult dimensions.

When such cases are encountered by the surgeon, particularly if the incision is made on the right side of the abdomen and the condition present is a right paraduodenal hernia the operative findings may appear as an apparent complete invisceration of the abdominal contents, since the colon is situated to the left side of the abdominal sac. The sac gives the impression of being extraperitoneal as far as the general peritoneal lining is concerned. The similarity between such invisceration and the better known paraduodenal herniae deserves mention.

Papez is also of the opinion that right and left paraduodenal (or retroperitoneal) herniae in the vast majority of instances probably arise in this manner and not as has been postulated by Moynihan and others by insinuation of the gut or inclusion under one of the paraduodenal fossae or folds. The small intestines in the majority of cases are the only structures enclosed in the special peritoneal sac which is suspended either from the transverse colon or the region of the duodenum, the former being the most common condition (left paraduodenal hernia). It seems that the right and left paraduodenal herniae and their surrounding sac have a more natural origin in the very early embryonic umbilical hernia. All told, about 34 cases of right and 105 cases of left paraduodenal herniae have been recorded. This interpretation assumes an identical origin for both right and left herniae from the early umbilical peritoneal sac. In case the large intestine has completed its rotation to the right of the sac, across the duodenum and mesenteric root before adhesion of the orifice of the sac and retraction of the gut has taken place, it would result in the more common form or left paraduodenal hernia, in which the abnormal sac is situated to the left of the ascending colon and is suspended from the transverse colon and its mesentery. In case the large intestine has not completed its rotation before adhesion of the orifice of the sac and retraction of the gut has taken place the condition produced would be a right paraduodenal hernia, in which the abnormal sac is situated to the right of the ascending colon.

The case reported by Doctor Goldsmith may reasonably be interpreted as a right paraduodenal hernia. In the more common left paraduodenal hernia the abnormal sac with the contained bowel lies between ascending and descending portions of the colon suspended above from the transverse colon.

PNEUMOCOCCUS PERITONITIS

AN ANALYSIS OF SEVEN CASES

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ABOUT 2 per cent of abdominal emergencies in childhood are due to pneumococcus peritonitis (McCartney and Frazer[11]). About 75 per cent of all cases of pneumococcus peritonitis occur in females under ten years of age (Blake[2]). About 0.24 per cent of cases of pneumonia develop pneumococcus peritonitis (Rolleston[15]). Most cases seen in girls are of the primary type. Most cases seen in boys are secondary in type. Less than one-third of all cases are secondary cases.

Pathology.—In the first stage of the disease there is no true pus. A thin, serous, turbid, odorless liquid, creamy or grayish yellow in color, is to be observed. Diffuse pus is formed in the intermediate stage. The peritoneum is not always markedly reddened. It has a slippery, slimy feel. A thick fibrinous exudate with the formation of adhesions and abscesses usually situated in the lower abdomen is encountered in the third stage. These abscesses may perforate spontaneously through the umbilicus. The third stage usually appears about the fourth day or thereafter. Coexistent pneumonia, pleurisy or pericarditis are common in cases of general pneumococcus infection.

Symptoms and Differential Diagnosis.—The symptoms may be violent or fulminating, with hyperpyrexia, cyanosis, rapid pulse and prostration. One of McCartney and Frazer's cases died within 36 hours. There may be excessive vomiting during the early acute stage which cannot be confused with the vomiting of obstruction because of its early appearance.

The facies are quite normal in the initial and progressive stage but Hippocratic facies may be seen in the third stage. This is a frequent sign. Pain is frequently complained of in the left lower quadrant instead of the right lower quadrant and midabdomen. Diarrhea is observed in the early stage, especially in the primary infections. It does not occur so frequently in the secondary type; males are more usually affected. The diarrhea is usually relieved. Tenesmus and blood-stained mucus sometimes occur. Frequent painful micturition may be distinctive.

Distention in the upper abdomen with tenderness and rigidity confined to the lower abdomen is the usual finding upon physical examination. Digital rectal examination should be made. The presence of an early mass may be determined.

Many authors have put a great deal of emphasis upon the fact that most cases present a high leukocytosis. The average count in our cases was

22,000. This is somewhat higher than the average count which one finds in acute appendicitis. Still, it is comparable to what might be expected in other types of peritonitis. Too much confidence, therefore, should not be placed in this finding.

One diagnostic point of great clinical value is the aspiration of the peritoneal cavity through a small needle. The diagnosis is rather definite if we find creamy odorless pus containing Gram-positive extracellular diplococci.

Prognosis.—Many writers are pessimistic regarding the prognosis. Rischbieth[14] found a mortality of approximately 90 per cent. McCartney and Frazer[11] report 65 per cent mortality. Later cases treated in their group in 1921 showed a reduction to 42 per cent. Ciminata[4] reported a mortality of 50 per cent in a group of eight cases.

Treatment.—The latest statistics show controversial opinions. The majority of authors favor late intervention. McCartney and Frazer advise early drainage subsequent to blood transfusions. They claim a reduction in the mortality from 65 to 42 per cent. Leipshutz and Lowenburg[8] likewise agree to early operation because they believe it is impossible to differentiate this type of peritonitis from other forms. Montgomery[12] and Schwartz also advocate early operation. Ombredanne,[13] who has had an extensive experience in infant surgery, believes operation should be done without delay.

Aurosseau,[1] Loewe,[10] Budd,[3] Davioud and Mathieu[5] have had a mortality varying between 75 to 95 per cent after premature intervention. Budde[3] and Salzer[16] have obtained a lowered mortality in the late stages of 6 to 25 per cent. Elkin[6] and Kahn[7] favor operation in the late stage. Lilenthal[9] believes these patients, like cases of gonorrheal peritonitis, should never be treated by operation. Ciminata[4] prefers to operate in the third stage. All of his cases were of the primary type. Two were operated upon in the first stage and two in the second stage. These four cases died. Four cases were operated upon in the third stage. All of them recovered.

Five of the seven cases herewith reported were of the secondary type. Three recovered. Operation had been delayed from nine days to several weeks after the onset of symptoms. The two cases of the secondary group which died were operated upon during the intermediate period. One of these had a perforated gangrenous appendix with a mixed infection of Gram-positive bacilli and pneumococci.

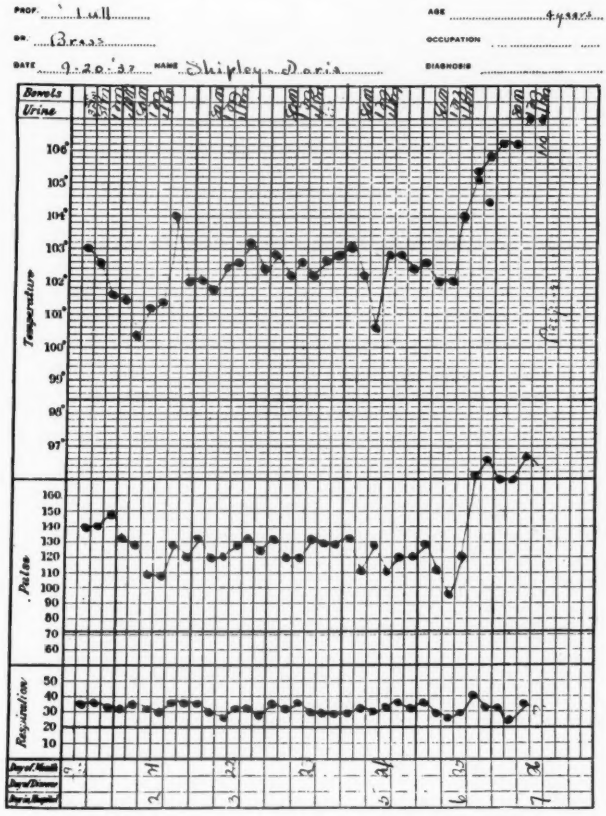
TABLE I

No.	Age	Sex	Duration of Illness Prior to Operation	Primary Infection	Result
1	32	M.	2 days	Acute grangrenous appendicitis	Died
2	15	F.	4 days	Bronchopneumonia	Died
3	9	F.	9 days	Bronchopneumonia	Cured
4	5	F.	14 days	Abscess of shoulder	Cured
5	5	M.	Several weeks	Bronchopneumonia	Cured
6	16	F.	7 days	Tuberculous peritonitis	Died
7	4	F.	6 days	Gonorrheal vaginitis	Died

Two cases of our group were of the primary type. One was operated upon six days after the onset of symptoms. The other patient, a colored girl, 16 years of age, had pneumococcus peritonitis complicating tuberculous peritonitis. She was operated upon seven days after the onset of acute symptoms (Table I).

The abdominal cavity was drained in each instance by abdominal section. Two patients were of sufficient interest to justify a short abstract of their record.

CASE VI.—F. B., colored, female, aged 16 years, was admitted to the University Hospital January 22, 1932. She gave a history of "feeling bad for one week." Acute,



GRAPH 1.—Showing range of T. P. R. in Case VII, indicating the advisability of waiting to operate late in these cases.

violent pain persisted for 24 hours prior to admission. The pain began during the act of micturition. There was no nausea or vomiting. The abdominal examination showed marked diffuse rigidity. She was more tender in the lower right quadrant. The tip of the cervix was soft and a boggy mass was found to the right of the uterus. The patient was operated upon January 23, 1932. A large pelvic abscess was drained through a midline incision under ether anesthesia. The patient died on February 5, 1932. An autopsy disclosed a disseminated tuberculous and pneumococcus infection of the peritoneum.

CASE VII.—D. S., white, female, aged four, was admitted to the Sydenham Hospital

June 28, 1933. The past history was positive for measles, chicken pox and whooping cough. The diagnosis was gonorrheal vaginitis. She was discharged July 24, 1933—condition improved. The patient reentered the Sydenham Hospital September 20, 1933. Temperature 103°, pulse 140 and respirations 35. A purgative had been given before admission. She pointed to the lower left quadrant when questioned about abdominal pain.

Physical examination showed marked abdominal distention with rigidity and tenderness in the lower half. Hippocratic facies were absent. The face was flushed but the child did not appear to be ill. Diarrhea persisted for several days. The blood cell count showed 17,500 leukocytes with 92 per cent polys. The following day 23,600 with 96 per cent polys. The radiogram showed a negative chest. A rather large mass developed in the midline of the lower abdomen five days after admission and vomiting recurred on that date.

Discussion.—I believed that the patient had been suffering from gonorrheal peritonitis notwithstanding the high cell counts. On September 25, six days after the onset of acute symptoms, the patient had a recurrence of vomiting. The diarrhea had subsided and enemata were effectual. It was decided to operate upon her and a small amount of odorless pus escaped from the right McBurney incision. No attempt was made to explore the abdomen or abscess cavity. Several drains were placed in the wound. The patient returned to the ward in good condition. She immediately became worse and died within 24 hours. Autopsy revealed pneumococcus peritonitis.

I think we would have been justified in aspirating the peritoneal cavity to prove the diagnosis in this case. The pulse rate (the best prognostic guide in peritonitis) appeared as though it had a tendency to subside on the date of operation. A better protective barrier might have been formed if the operation should have been delayed several days longer. We believe the course of the charted pulse rate is a valuable subsidiary index which may be used to determine the proper time for operation.

Operative intervention prior to the formation of encysted peritonitis will lead to disaster and continued high mortality in this condition.

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PENETRATING WOUNDS OF THE ABDOMEN*

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THIS analysis of penetrating stab and gunshot wounds of the abdomen comprises 164 cases treated at the Harlem Hospital, New York, from March, 1930, to January 1, 1935, under the direction of the late Dr. John Fox Connors.

In comparison with other hospitals in New York City, Harlem Hospital receives many of these cases. Because of a breakdown and change in system in the record room, it was necessary to start this present series in March, 1930.

There were 100 cases of penetrating stab wounds of the abdomen with 21 deaths, a mortality of 21 per cent. There were 64 cases of penetrating gunshot wounds of the abdomen with 38 deaths, a mortality of 59.3 per cent. The total number of deaths in the two series was 59, a combined mortality of 35.9 per cent.

The two groups are considered separately, because the stab wounds on the whole are less serious than the gunshot cases, by reason of the more limited range of the knife blade. In the gunshot cases there is apt to be more hemorrhage and more extensive visceral injury.

In this series, as in others, the usual ratio of the greater severity of the gunshot over the stab injuries was maintained, that is, a little more than two to one. A brief review of the published statistics will show that the operative mortality in civil life seems to have been established around 50 per cent for gunshot wounds, and around 25 per cent for penetrating stab-wound injuries.

The New Orleans Charity Hospital statistics on penetrating abdominal gunshot injuries show 1,299 cases as having been treated at that institution in 32 years (1900–1931), with a mortality of 62.3 per cent.

Wallace,[1] reporting 1,200 gunshot cases from the British Expeditionary Force, showed an operative mortality of 53.9 per cent, and a total mortality, including nonoperated cases, of 60.2 per cent. Lockwood, Kennedy, *et al.*,[2] in military service, reported 500 cases with an operative mortality of 51.9 per cent. Mason[4] analyzed 127 cases and emphasized the influence of hemorrhage on mortality, dividing his series into large and small hemorrhage groups, regardless of visceral injury, with a mortality in the large hemorrhage group of 87.2 per cent, and one of 36.1 per cent in the small hemorrhage group.

Billings and Walkling,[6] in a comprehensive study of 220 cases of penetrating wounds of the abdomen admitted to the Pennsylvania Hospital

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over a period of 21 years, reported an operative mortality of 22 per cent in stab wounds and 48 per cent in gunshot wounds. These writers, and also Winslow,[3] in a report of 31 cases treated at the University Hospital in Baltimore with 16 recoveries and 15 deaths, noted the importance of injuries overlooked at the time of operation. More recently, Prey and Foster[7] have reported 22 cases of gunshot wounds admitted to the Denver General Hospital, from 1928 to 1933, with a mortality of 68 per cent.

These patients as a group are usually active, robust individuals, the greatest number being between the ages of 20 and 40. In this series of 164 cases, 129 were in this age group. Most writers have pointed to the high proportion of colored patients in their series. In this group of 164 cases, there were 127 colored patients (Table I). While not noted in the individual cases, alcoholic intoxication was an accompanying factor in many patients.

TABLE I

PENETRATING WOUNDS OF THE ABDOMEN

From March, 1930, through December, 1934

Total number..... 164
Mortality..... 59, or 35.9 per cent

Stab Wounds—100 cases

91 males..... 85 were colored
9 females

There were 21 deaths in the series, or 21 per cent

Gunshot Wounds of Abdomen 64 cases

57 males..... 42 were colored
7 females

There were 38 deaths in this group, or 59.3 per cent

AGES

	Gunshot	Stab
1–10	0	0
10–20	7	2
20–30	26	45
30–40	23	35
40–50	6	13
50–60	1	5
60–70	1	

In 79 surviving penetrating stab wounds of the abdomen, there were 16 cases of penetration of the abdominal wall and parietal peritoneum but no visceral injury. Subtracting these cases, we have 63 cases with 21 deaths, or a revised mortality of 33.3 per cent.

It seems to the writer that a more accurate comparison between the severity of stab and bullet wounds may be made if the above type of case is not considered in figuring the mortality of stab wounds. True, they come under the classification of penetrating wounds but they are not accompanied

by any visceral injury—a happening not uncommon in stab wounds but quite unusual in bullet wounds of the abdomen.

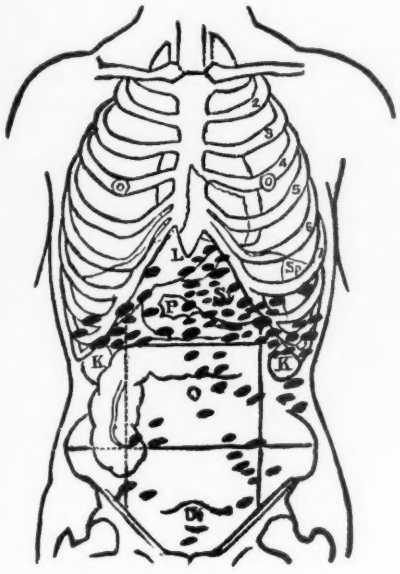

FIG. 1.—Location of stab wounds (surviving).

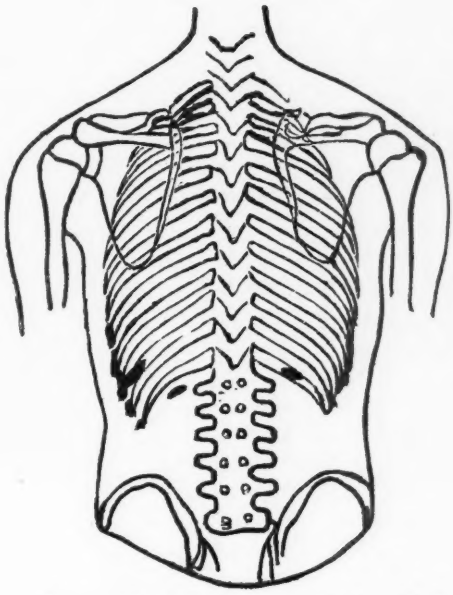

FIG. 2.—Location of stab wounds (surviving).

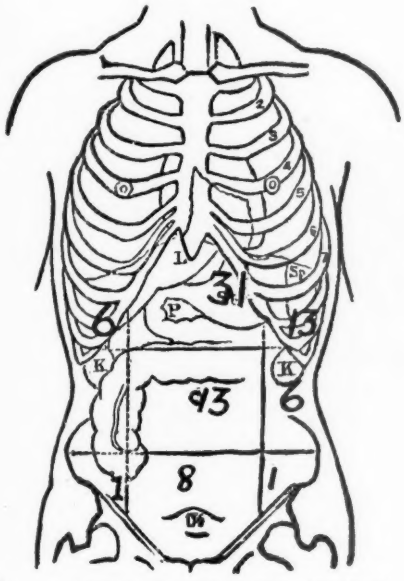

FIG. 3.—Location of stab wounds (surviving).

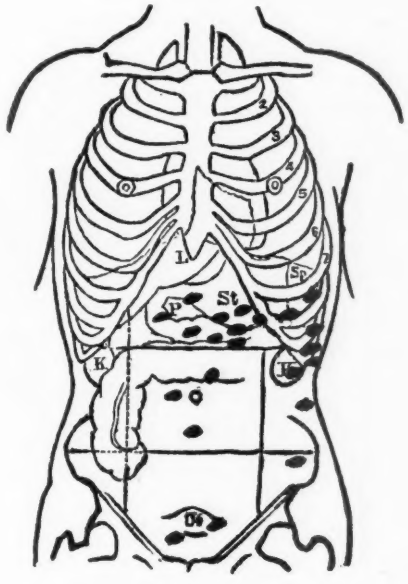

FIG. 4.—Location of stab wounds (fatal cases).

There were 21 cases presenting penetrating stab wounds of the abdomen with evisceration of omentum. In nine of these the injury was confined to the omentum (Table II).

TABLE II

Cases of Evisceration of Omentum with Associated Injuries

	Liver	Costophrenic Sinus	Small Intestine	Stomach	Diaphragm	Retroperitoneal Hematoma
1.	X	X			X	
2.			X (2)			
3.				X		
4.	X					
5.				X (serosa)		
6.			X			
7.				X		
8.			X			
9.				X		
10.		X		X (serosa)	X	
11.				X		
12.			X (2)			X

There were 17 cases of penetrating stab wounds with evisceration of major degree (Table III).

TABLE III

Injuries

Evisceration (17 cases)	Stomach	Spleen	Small Intestine	Large Intestine	Diaphragm	Liver	Retroperitoneal Hematoma
1. Omentum, small intest.		X					
2. Omentum, small intest.			X				
3. Greater part of small and large intest.			X (5)				
4. Stomach trans. colon, omentum	X (serosa)						
5. Loop of small intest.							
6. Complete evisceration			X (serosa)				
7. Complete evisceration							
8. Small intestine			X (serosa)				
9. Colon, spleen, omentum				X (2)	X		
10. Small intestine			X (6)				
11. Trans. colon, omentum, small intest.						X	
12. Stomach, omentum	X						
13. Small intestine							

TABLE III—(*Continued*)

14. Small intestine, omentum				
15. Small intestine	Loop strangulated			
16. Hepatic flex., colon, small int.				
17. Small intest.		X (serosa)		X

There were 25 stab wounds (1 case not operated upon) without evisceration but with the following intra-abdominal injuries (Table IV).

TABLE IV

Injuries in Cases of Penetrating Stab Wounds without Evisceration

	Blood	Liver	Stomach	Spleen	Gallbladder	Small Intestine	Retroperitoneal Hematoma
1.	++++	X					
2.	+++	X					
3.			X (serosa)				
4.		X					
5.	++++		X (2)	X			
6.					X (2)		
7.						X	
8.			X				
9.	+++	X					
10.	+						X
11.	+		X (serosa)				
12.	++	X					
13.	++		X (serosa)				
14.							X
15.	++	X					
16.		X					
17.	++	X					
18.							X
19.		X					
20.			X				
21.						X	
22.			X				
23.	+++	X					
24.		X					

It is important to note that in this group of 79 surviving cases of penetrating stab wounds of the abdomen, only five cases presented retroperitoneal hematomata, one case presented a double penetrating wound of the large intestine (anterior wall) and one case presented a serosal wound of the large intestine.

There were no cases of injury to the duodenum, pancreas, extraperitoneal portion of large intestine or rectum. No case presented extensive retroperitoneal hemorrhage. Outside of controlling hemorrhage by packing (in liver wounds) there was, in general, no indication for intraperitoneal drainage in this series.

TABLE V

Fatal Stab Wound Cases

Evisceration	Blood	Large Intestine	Small Intestine	Stomach	Liver	Costophrenic Sinus	Spleen, *etc.*	Dia-phragm	Retroperitoneal Hematoma
1. Bleeding omen.	+++								
2. Omentum	++++	X	X						
3.	+++	X							
4. Omentum				X					
5.	++++				X				
6.	++++					X	X		
7. Omentum				X (2)			(Ass. head injury)		
8.	+++		X (serosa)	X (serosa)					
9. Omentum		X (2)		X					
10. Colon						X		X	
11. Omentum			X (2)						
12.	+++			X (serosa)					
13.	+			X					
14. Omentum				X (2)					X (P. M. R. R. artery cut across)
15.						X		X	X
16. Small intest.			X						
17. Trans. colon, small intest. omentum				X					
18.	+++		X (6)				(Ass. chest inj.)		P. M. Left hemothorax
19.						X	(Ass. chest inj.)	X	P. M. Rt. hemothorax (diaph. and liver)
20. Half of gastro-intestinal tract, omentum			X (2)						
21. Small intest.	+++	X	X (3)						

We can readily group the causes of death in the cases of fatal penetrating stab wounds as follows: hemorrhage, 12 cases; suppurative peri-

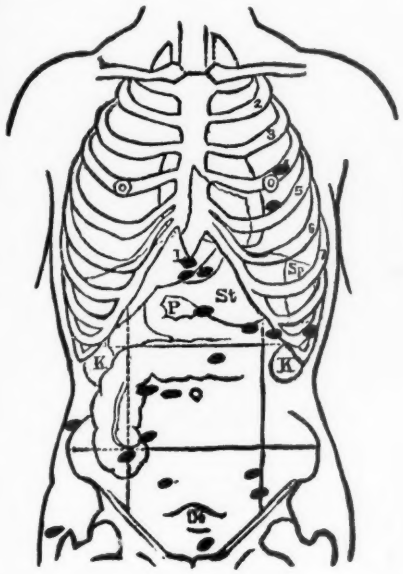

FIG. 5.—Bullet wounds of entrance (surviving).

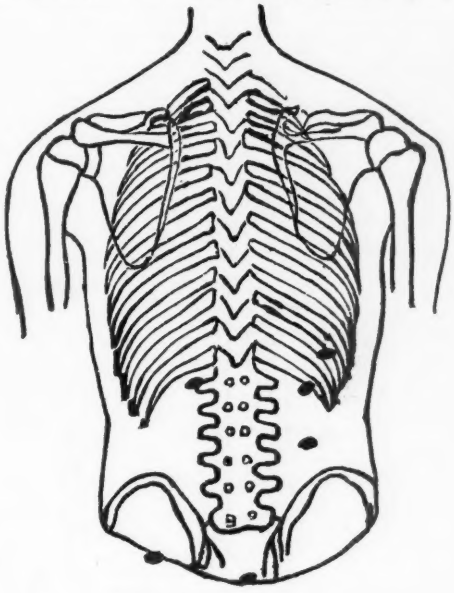

FIG. 6.—Bullet wounds of entrance (surviving).

tonitis, five cases; intestinal obstruction (with volvulus), one case; acute pneumonia, two cases; pylephlebitis (? liver abscess), one case. Thus in

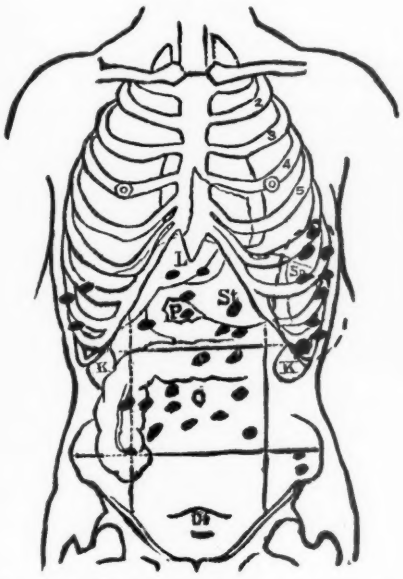

FIG. 7.—Bullet wounds of entrance (fatal cases).

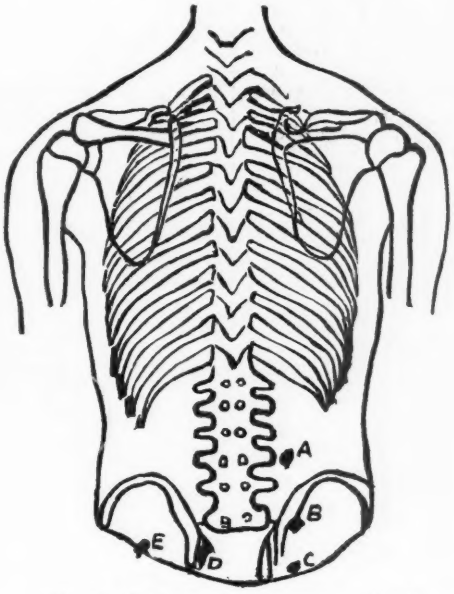

FIG. 8.—Bullet wounds of entrance (fatal).

17 cases, or 80.9 per cent of total deaths, it was due to hemorrhage in 12 and suppurative peritonitis in five instances. Nine cases died within 24

TABLE VIII

Lesions in Surviving Gunshot Cases

	Time in Minutes from Injury to Operation	Shock	Blood	Liver	Stomach	Gallbladder	Small Intest. No. of Perforations	Large Intest.	Omentum	Rectum	Bladder	Kidney	Spleen	Retroperitoneal Hematoma	Associated Injuries
1.	120	+	+++	+	+	+ (2)									
2.	60	+	++++		+ serosa										
3.	330	0	++						+						
4.	300	0						+						+	
5.	360		++											+	
6.	120		++	+		+									
7.										Meso					
8.	90	0	+	+											
9.	180	0	+++	+											
10.	180		+					Serosa						+	
11.	60		+++		+ (2)									+	Chest wall pleura, diaph.
12.	480	+	++++	+											
13.	105		0		+ (2)		+								
14.		0					+			+ (2)	+				
15.	120		++++				+ (11)								
16.	180		++		+										
17.	120		++	+				+ (3)							
18.	180	0	++				+ (2)			+ (2)	+ (2)				
19.	165	+	++++										+		Lt. diaphragm
20.	150	+	+				+	+ (4)						+	
21.	60	+	++	+					+						
22.	150		++++				+ (9)								
23.	120	0	0				+ (8)	+ (4)						+	
24.	480		++++											+	
25.	150		+++				+ (9)								
26.	1020		++	+											

hours, five within the first week, and the seven others ranged from 13 to 43 days (Table V).

There were a total of 64 cases of penetrating gunshot wounds of the abdomen with 38 deaths, or a mortality of 59.3 per cent.

Surviving Cases.—Time elapsing between injury and exploration 1 hour to 17 hours, an average time of 3.6 hours. Definite shock was noted in six cases on admission. When the amount of hemorrhage was noted, it was estimated as large in 10 cases and moderate in 11 cases.

TABLE VI

Penetrating Bullet Wounds of Abdomen

	Surviving Cases 26	Fatal Cases 38
Time elapsing bet. injury and exploration	3.6 hours	3.7 hours
Clinical shock was noted as present on admission in —	6 cases, or 23%	31 cases, or 81.5%
When the amount of hemorrhage was noted it was noted as —	large in 10 cases, or 38.4% moderate in 11 cases, or 42.3%	large in 23 cases, or 60.5% moderate in 7 cases, or 18.4%

TABLE VII

Visceral Lesions

Liver	8	Small intestine	8
Retroperitoneal injury (hemor.)	7	Omentum	2
Stomach	5	Diaphragm	2
Large intestine	5	Spleen	1
Rectum	3	Pleura	1
Urinary bladder	2	Gallbladder	2

Analysis of Fatal Cases.—In 38 cases, autopsies were done on 27 (71 per cent). Five cases died on the operating table (autopsies on two of these). Time elapsing between injury and exploration, one hour to ten hours, with an average time of 3.7 hours. Excluding the five cases that died on the operating table, the survival times ranged from one to 13 days postoperative, with an average survival of 3.7 days. Definite clinical shock or collapse was noted in 31 cases on admission. When the amount of hemorrhage was noted, it was estimated as large in 23 cases and moderate in 7 cases. Lesions were missed in 15 cases (Table IX).

TABLE IX

Lesions Overlooked

Liver	4 times	Pancreas	2 times
Large intestine	2 times	Pleura	2 times
Rectum	2 times	Diaphragm	3 times
Kidney	3 times	Lung	1 time
Duodenum	1 time	Adrenal	1 time
Spleen	2 times	Inf. vena cava	2 times
Retroperitoneal hemorr.	2 times	Portal vein	1 time
		Iliac vein	1 time

TABLE X

Visceral Lesions in 38 Fatal Cases

Small intestine	21	Pancreas	5
Large intestine	20	Lung	3
Retroperitoneal injury, hemorrhage	19	Spleen	3
Liver	11	Inf. vena cava	2
Stomach	12	Iliac vessels	2
Pleura	6	Gallbladder	2
Duodenum	6	Urinary bladder	2
Omentum	5	Rectum	2
Kidney	5	Adrenal	1
		Portal vein	1

In 38 gunshot deaths following operation, there were autopsy reports on 27 cases. These findings were made available for study through the cooperation of Dr. Charles Norris, the chief medical examiner of New York City. The records showed that in 15 cases, visceral injuries had been overlooked at the time of operation. This checking of the operation against the postmortem findings will show that the occurrence of overlooked injuries is more common than is usually supposed. Those who have had experience in dealing with cases presenting a multiplicity of visceral injuries will appreciate the many difficulties encountered at operation.

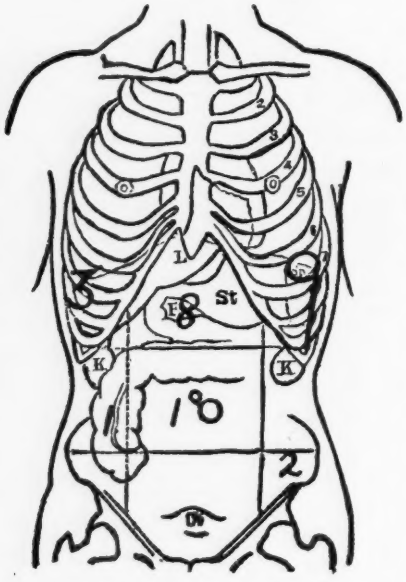

FIG. 9.—Bullet wounds of entrance (fatal cases).

Billings and Walkling,[6] in their survey reporting 55 gunshot operative deaths with autopsy examination on 49 cases, found nine instances in which visceral injuries of one kind or another had been overlooked at the time of operation.

The average time between receipt of injury and surgical exploration was 3.7 hours. The importance of shortening this time interval has been stressed by others but we feel that time intelligently spent in the treatment of the patient's initial shock is time well spent and that one may do harm by rushing a patient in shock to exploration.

At the Harlem Hospital, a definite plan of management of these cases has been instituted. Immediately after admission, treatment for shock is instituted; that is, a continuous intravenous drip of 5 per cent glucose in saline, external heat, and morphine. Blood typing is done and in cases with lower abdominal bullet tracts the bladder is catheterized. The blood pressure is followed carefully. Most of the patients are radiographed on their way to the ward or operating room with a minimum of handling.

Abdominal and chest roentgenograms are frequently invaluable in indicating the not infrequent bizarre course of the bullet. The associate surgeon on call is notified immediately. Measures to combat shock are continued and the majority of our serious cases have their intravenous drip started on admission and continued without interruption until their third or fourth postoperative day.

The great importance of blood transfusion, before, during, or after operation, has been frequently stressed by many writers on this subject and is emphasized again here. At the Harlem Hospital in recent years, we have been fortunate in being able to transfuse patients without undue delay. A patient may come to exploration in apparently good condition and suddenly go into collapse and die. Surgeons dealing with this type of case are familiar with this sudden sequence of events and it remains a possibility to be constantly borne in mind and guarded against by the provision of blood donors and their more frequent use.

The choice of anesthesia is an important one. The majority in this series were given open ether. The writer has employed, in selected cases, spinal anesthesia guarded by a continuous intravenous drip and frequent doses of ephedrin with satisfactory results.

Three factors operate in determining the gravity of gunshot wounds: (1) most important, the degree of visceral damage; (2) the severity of the hemorrhage; and (3) the time elapsing between the receipt of injury and surgical intervention. The latter two factors have been touched upon above. The first factor demands more consideration. I do not believe that the importance and gravity of injuries to the retroperitoneal tissues have been sufficiently emphasized. The retroperitoneal tissues may be injured directly, or indirectly by penetration of the duodenum, pancreas, retroperitoneal portions of the large intestine, and rectum.

We see repeatedly cases of injury to a solid viscus or a peritonealized hollow viscus which survive although the lesions may be multiple, but in cases presenting penetration of the duodenum, pancreas, extraperitoneal portions of the large intestine, and the rectum, our prognosis is necessarily guarded because of the accompanying laceration of the retroperitoneal tissues, and their tendency to bleed and undergo suppuration and phlegmonous infiltration.

The technical procedures in our surgical repair should be comprehensive but as simplified as possible. Tamponade of a lacerated bleeding wound of the liver is adequate, whereas suture is tedious and uncertain. Multiple enterorrhaphy is preferable to resection, with proximal enterostomy as an aid when repair has been difficult. Extensive lacerations of the large intestine may be speedily handled by exteriorizing the injured portion. Laceration of the spleen demands splenectomy. Injuries to the duodenum and pancreas should be thoroughly visualized. The duodenum should be mobilized, perforations closed, and the adjacent retroperitoneal space drained. This is most important in pancreatic injuries where wide

TABLE XI

Résumé of Analysis of Fatal Cases

	Liver	Stomach	Small Intest.	Large Intest.	Omentum	Rectum	Bladder	Kidney	Duodenum	Gallbladder	Retroperi. Hematoma	Pleura	Diaphragm	Lung	Adrenal	Shock	Hem.	Time	Lived		Pancreas	Spleen
† 1.			+ (3)	+*								+	+			+	++++	105	10	G. P.		
† 2.	+	+ (2)						+			+		+			+	+++	120	3	G. P.		
† 3.	+*			+	+						+		+	+				180	3	G. P.	+*	
† 4.				+ (2)				+			+					+	++++	120	13	G. P.		
† 5.		+ (2)	+ (6)	+ serosa	+			+*				+	+	+		+	++++	180	2	Chest		+
† 6.			+ (6)	+													++++	180	2	G. P.		
7.			+ (13)				+										+	150	9	G. P.		
† 8.	+*	+							+		+					+	++++	120	1	Hem.		Vena cava*
9.		+ (2)							+		+					+		60	O. T.		+	
†10.			+	+ (2)												+	++++	70	1	Hem.		
†11.			+	+							Sept. +					+		120	2	G. P.		
12.		+ (2)									+					+	++++	270	O. T.	Hem.	+	
†13.				++++				+*			+*		+*			+	++	180	1	Hem.		+*
†14.	+	+			+							+	+			+	+++	120	5	G. P.		
15.	+	+2	+3	+					+							+	+++	90	O. T.	Hem.		
16.			+													+		60	2			
†17.				2*							+					0	++	120	5	G. P.		
				+2																		

†18.			+6	+ meso.							+				+	++	120	7	G. P.		
19.			+6								+					++++	150	1			
20.			+ mesent.	++							+				+	++++	210	1	Hem.		
21.				+ serosa							+						600				
†22.	+						+*					+*	+*		+	++++	90	O. T.	Hem.		
†23.			+ (10)	+											+	++++	180	3	G. P.		Rt. iliac vessels
24.		+ (2)	+ (4)	+ (4)						+ (2)					+	++++	60	3	G. P.		
†25.	+				+						+ *	+*	+*	+*	+	++++	210	2	Hem.		Portal V*
†26.			+ 8			meso.									+	+++	120	6	G. P.		
						+*															
27.		+ 2													+	+	105	6	G. P.	+	
†28.			+2+												+	++++	60	O. T.	H.		+*
29.		+													+		240	6	Chest		
†30.			+ 15			+*									+	++	180	6	G. P.		
†31.				+ 2					+	+	+				0	+++	120	1			
†32.	+*		+ 5	+ 2	+				+*		+				+	++++	90	2	G. P.		
†33.		+ 2										+	+	+*	+	+++	90	2	Chest		
†34.			+ 12	+ 2			+ 2				+				+	++++	90	4			Iliac V.*
†35.	+*	+	+ 9								+				+	++	150	1	H.	+*	Vena cava*
																			G. P.		
†36.	+		++++												+	++++	180	2	G. P.		
†37.			+ 8	+							+				+		60	6	G. P.		
†38.	+			+					+						+		180	1	G. P.		

Note: †=Autopsy cases.

+*=Overlooked lesions.

drainage is essential. The significance of the posterior perforation accompanying the anterior perforation in a peritonealized hollow viscus is generally appreciated, but in the fixed portions of the large intestine and especially the rectum, it is of great importance. Posterior wounds of the ascending and descending colon demand mobilization of the injured portion, careful closure of the perforation, and wide drainage to the adjacent retroperitoneal space. Injuries to the rectum, in addition to the intraperitoneal repair, demand excision of the coccyx and exploration of the retrorectal space with closure of the posterior wall perforation and the institution of wide drainage to the retrorectal space.

A large retroperitoneal hematoma had better be left untouched unless it rapidly increases in size. These are grave injuries and exploration is apt to be futile and disastrous.

To facilitate intra-abdominal exploration and repair, we should not hesitate to use long incisions, and to deliberately eviscerate the patient. The eviscerated contents are carefully protected with large, warm, moist packs. I believe that heat applied in this direct manner to the entire splanchnic area, which frequently feels definitely cool to the hand, is of distinct aid in combating shock. In addition, the procedure is of the utmost value in aiding our search for injuries, expediting their repair, minimizing the incidence of overlooked injuries and materially shortening the operating time.

CONCLUSIONS

(1) Penetrating wounds of the abdomen remain one of the most difficult problems in the surgery of the acute abdomen.
(2) Penetrating wounds inflicted with the knife are less dangerous than when inflicted with the bullet.
(3) Blood transfusions are invaluable.
(4) Attention is called to the importance of adequate exposure—obtained by long incision and evisceration.
(5) The gravity of retroperitoneal injuries with their demand for wide exposure and extensive drainage is emphasized.

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DISCUSSION.—DR. EUGENE H. POOL (New York) recalled a considerable experience with such accidents in the old Hudson Street Hospital where Doctor Bull did his first suture of a wound of the intestine. There were many Chinese patients as a result of tong warfare. A Chinaman puts a 44 gun under his left arm, marches up to his victim, puts his elbow against the victim's abdomen and pulls the trigger. As a rule the bullet passes through the stomach. A relatively small orifice in the anterior wall of the stomach was usually found, but a vast lacerated wound in the posterior wall. As emphasized by Doctor McGowan, the retroperitoneal structures were variously involved and this was often the important factor. Few survived.

During the War many cases were seen. Débridement was shown to be of primary importance. A recent case brought out a point to which Doctor McGowan did not refer: that is, the possibility of the development of gas-bacillus infection as the result of penetrating wounds, but not resulting from the projectile. If the projectile shatters a bone, bony fragments may become contaminated as the result of passing through the intestine, and lead to gas-bacillus infection. There was one such case in the old New York Hospital. A man was accidentally shot through the sacrum. There were perforations in the large bowel, loops of small intestine, and the bladder. All of these wounds were successfully repaired, but after 36 hours the patient developed a peculiar condition which should have been recognized but was not, and proved to be a gas-bacillus infection of the abdominal wall. Autopsy showed that the peritoneal cavity was clean and that the wounds had been properly repaired, but the bony fragments from the sacrum which had been projected as independent projectiles through the large intestine had been carried into the muscle of the abdominal wall; they had carried microörganisms which were deposited in the devitalized muscle along the tracts of the fragments in which they found a proper medium for their growth. In that case, if proper therapeutic measures had been taken, the patient might have been saved.

DR. JOHN E. JENNINGS (Brooklyn), in searching the records at the Cumberland Hospital in Brooklyn for the past three years, found 30 cases of penetrating wounds of the abdomen. Fourteen of these were gunshot wounds, in which the mortality was practically the same as in Doctor McGowan's series, i.e., 50 per cent. In the stab wounds, the mortality was 33 per cent. A number of the cases (six or seven) of stab wounds of the abdominal wall had been conservatively treated, without opening the abdomen, and all recovered. In looking over the first four volumes of the ANNALS OF SURGERY and the ten volumes of the Annals of Anatomy and Surgery immediately preceding it, covering the time when the treatment of gunshot wounds of the abdomen by laparotomy was an innovation and a matter to be discussed, and especially in looking over the records of Doctors Bull and Andrews, it was interesting to note that Andrews twice reported four cases in which three recovered without operation. At the same time Doctor Bull began exploring them and reported his success. After making this survey, Doctor Jennings concluded that if the reports of the history of surgery the world over were gathered together and a comparison made of the damage done to the abdomen by gunshot fire with the results of this era, no tremendous advance would be noted. He suggested it would be most interesting if a study of the literature be made, not so much statistically as critically, to find out why the patient died. In a survey of 14 cases of gun-

shot wounds in Cumberland Hospital—seven of whom recovered, seven of whom died—the following facts stood out: In the cases who lived, the operating time was a trifle under one hour; in those who died, something over two hours; inasmuch as the same hands operated in these cases, in all probability the difference in operative time is a fair index of severity of the injury. Cases also did better under general than under spinal anesthesia. Moreover, the degree of manipulation within the abdomen, not only the time spent but the degree with which evisceration occurred, bears a direct relation to mortality. Doctor Andrews—going back to 1888—said he thought it wise to explore the abdomen as soon as necessary to detect, as far as possible, the amount of hemorrhage and as far as possible the amount of leakage and proceed according to what one found at this exploratory operation.

Dr. Howard Lilienthal referred to his experience during the World War and emphasized one point in particular, namely, when a patient has been wounded through the abdomen and there is thoracic injury as well, after the abdomen has been attended to, the chest should be opened by a wide intercostal incision and examination made in that way using a rib spreader. The incison does not take long and is not shocking if done in that way. Before the wound of the diaphragm is repaired, the phrenic nerve as it runs through the pericardium should be found and given a pinch with forceps. This makes the operation much easier and relieves the danger of the reopening of the diaphragmatic wound. When this muscle contracts with respiratory effort the recently sutured wound is liable to reopen.

Dr. Herbert Willy Meyer referred to a case he presented seven years ago before the Surgical Section of the New York Academy of Medicine. The patient was a young gangster who was shot from behind, receiving two bullet wounds over the sacrum, then at close range he was shot three times more. He was admitted to the Lenox Hill Hospital with five bullet wounds of the soft lead type. One was close to the cervical vertebra, on the first rib. A second bullet was in the soft parts in the axilla. Two other bullets were over the sacral region, and a fifth near the first lumbar vertebra. The fifth bullet had passed through the twelfth rib. It could be felt under the skin, and had entered through the ninth interspace, in the axillary line, passing through the costophrenic space and abdomen and out through the twelfth rib. As we observed him his poly count went up rapidly with the white cell count. The left side of the abdomen became rigid.

A left rectus incision was made and a large amount of blood encountered in the abdomen. An injury of the spleen was felt. The patient had a very high costal arch. An additional transverse incision was made in an attempt to get up to the spleen which lay high under the diaphragm and was very small. It was impossible to get at the pedicle. There was profuse hemorrhage. An attempt was made to get better access by a chondroplastic resection of the costal arch. A flap was turned up between the rectus muscle and the costal arch. The costal cartilages near the sternum and at the juncture with the ribs were divided. With the posterior rectus sheath and the costal cartilages turned up and back, an excellent access to the spleen, diaphragm and stomach was obtained. The spleen was removed. The wound in the diaphragm, now visible, was stitched, a drain put in, and the wound sutured. The patient had a somewhat stormy convalescence, developing an effusion in the chest which was aspirated and did not become infected. He had a *Staphylococcus albus* infection of the wound. Later when the

bullet was removed from the neck a *Staphylococcus albus* infection was also found around it.

Doctor Meyer called attention especially to this procedure as having been published many years ago by Marwedel abroad and presented by his father many times before the New York Surgical Society. In difficult cases—not only of gunshot wounds—under the vault of the diaphragm it provides a rapid and excellent access to the under surface of the diaphragm, the costal cartilage, heals very rapidly and gives a firm wound.

DR. F. W. BANCROFT (New York) called attention to one other type of penetrating wound of the abdomen which, in his opinion, should be treated in a different way. He referred to abdominal birdshot wounds, apt to be encountered in the hunting season. At the Southern Surgical Association meeting last December a paper was read on the subject, wherein it was definitely shown that the best results are obtained by not operating immediately. The shots used for bird shooting are small and when they penetrate the intestine are apt to seal over. Therefore, if no large vessel is injured, the peritoneum protects itself and the patient is likely to recover. These patients are usually in shock and if immediate operation is performed the mortality is fairly high. They should, therefore, be treated first for shock, with careful observation of the blood pressure. If the pressure tends to go down and the pulse up it is a sign that hemorrhage is present and therefore it is advisable to operate. If infection does occur, it will probably form a localized abscess which may be drained at a later time.

Regarding war surgery, he felt that the advance made since the time of the Civil War was not always realized, and cited the interesting case of General Nelson A. Miles, whom he had had as a patient 15 years ago in New York Hospital. General Miles had a scar on his abdomen and when asked where he had received it answered that it was at the battle of Antietam. He was 25 years old at the time, but was already a brigadier general in charge of an army corps.

"I was on my horse," recounted the General, "when I saw a sharpshooter rise up. I turned my horse back, felt a bullet hit me in the region of the abdomen and thought I was paralyzed below the waist. I managed to stick on my horse, however, and got back to brigade headquarters. About that time a shell came along and destroyed all of the brigade headquarters except the area where my orderly and I were. I was kept there for two days, expecting that peritonitis would set in. When this did not occur, I was moved back eight miles over a corduroy road to a field hospital, where I remained five days in the expectation that I would die of peritonitis. As this did not transpire, I was later moved back to Norfolk and, as still my demise did not take place, was transported by ship to Washington. In the hospital there, a consultation was held. The surgeon said, 'Evidently you have not peritonitis at present, or you would not be here; therefore it is safe to probe your wound.' They found that the bullet had been deflected by my gold buckle and had shattered the ilium. They took out several pieces of dead bone but could not find the bullet. Finally, the chief surgeon inserted a long clamp into my thigh, found the bullet, and, placing his foot against the bed, heaved and pulled until he was able to remove it. After he had removed the bullet, I asked him what would be the result. He answered, 'It will be all right unless mortification sets in.' I said, 'What will you do then?' to which he replied, 'We will give you opium enough for the end.' "

REGIONAL (TERMINAL) ILEITIS*

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IN 1932, Crohn, Ginzburg and Oppenheimer[1] segregated from the miscellaneous group of benign granulomas a necrotizing and cicatrizing inflammation confined to the terminal ileum which they believed to be a pathologic and clinical entity, and called it "regional ileitis." Their report is based upon the study of 13 resected specimens. In the fully developed case the 8 to 12 terminal inches of the ileum, ending abruptly at the valve of Bauhin, is thick, boggy and edematous. The serosa is a blotchy red color and the mesentery of the involved segment is thickened and fibrotic and contains numerous hyperplastic nodes. The lesion apparently begins at the valve and gradually tapers off as it ascends the ileum orally. They describe oval mucosal ulcers about one centimeter in diameter occupying the long axis of the mesenteric border which they feel may be the primary lesion. The mucosa is the seat of a diffuse destructive ulcerative process. The remaining coats of the bowel are markedly thickened from inflammatory hyperplasia, giving the involved segment a hose-like structure. The fully developed case shows a marked stenosis of the lumen and, frequently, walled off abscesses and perforations, with resultant fistulae which may extend into the colon. Perforation into the mesentery with the development of a walled off abscess may give rise to pericecal and periappendiceal adhesions and thickening, giving the roentgenologic appearance of hyperplastic tuberculosis. Although focal inflammatory lesions of the serosa resembling tubercles occur, animal inoculations in five of Crohn's cases were negative, and Lowenstein found negative cultures for tuberculosis in three of his cases, in which there was no evidence of pulmonary tuberculosis. Giant cells were present in some cases which they attributed to resultant foreign body reaction around nonabsorbable vegetable foreign particles. These authors offer no explanation concerning the etiology or pathogenesis of the disease. Crohn[2] feels that this clinical entity is not rare since he has seen 28 additional cases in the two years which have elapsed since the original report. Young adults are usually affected.

The clinical features resemble chronic ulcerative colitis; diarrhea (but without tenesmus and usually little blood), lower abdominal pain, fever, continuous loss of weight and anemia. As the lesion begins to obstruct, attacks of abdominal pain (usually in the lower right quadrant), increased peristalsis, and often vomiting, occur. Fistulous tracts may be responsible for local-

* Read before the Joint Meeting of the New York Surgical Society and the Philadelphia Academy of Surgery, February, 1935.

izing symptoms elsewhere in the abdomen. The outstanding physical findings according to the authors are: (1) a mass in the right iliac region; (2) evidences of fistula formation; (3) emaciation and anemia; (4) a scar of a previous appendectomy (half the cases), and (5) evidence of intestinal obstruction.

The roentgenographic findings show obstruction of the terminal ileum without evidence of an intrinsic lesion in the cecum except for a possible fistulous tract. Kantor[3] has made a real contribution in his roentgenologic study of six personally observed cases and a review of the material of others. He described (*a*) "the string sign" in the terminal ileum, which is similar to that originally described by Crane as occurring in the colon in mucous colitis; (*b*) filling defects proximal to the cecum; (*c*) an abnormal contour of the last filled loop of ileum, and (*d*) dilatation of ileal loops proximal to the obstruction. He likewise mentions reflex colonic spasm with contractures of the cecocolon but more likely of the cecum proper (teat-like). An actual defect on the mesial aspect of the cecum may be due to adhesions or fistula from the diseased ileum.

Case Report.—A. L., Jewish multipara, 28 years of age, came to the Gastro-Intestinal Clinic of the Graduate Hospital, September 14, 1933, complaining of diarrhea of three months' duration and lower abdominal pain of two months' duration. The past medical history was irrelevant. The pain occurred in irregularly spaced attacks, was griping and crampy in character, localized largely to the region of the umbilicus and lower right quadrant of the abdomen. It was not related definitely to the ingestion of food but was relieved temporarily by defecation. The diarrhea was intermittent but she often passed from four to seven mushy stools daily, containing mucus. There was no complaint of tenesmus or local rectal discomfort. Vomiting occurred very occasionally. She had lost about ten pounds during the previous year. The original weight before the illness was 160 pounds. A tentative diagnosis of ulcerative colitis, possible minimal tuberculous lesion at the apex of the right lung and incipient ileocecal tuberculosis was entertained. These impressions were not confirmed by laboratory studies. Barium enema and sigmoidoscopic examination showed no evidence which could be interpreted as indicative of organic disease in the colon. The chest film was negative for acid-fast pathology. No ova or parasites could be found in the stools. She was placed on a smooth bland diet and given an antispasmodic sedative mixture.

She was admitted to the hospital August 14, 1934, about 14 months after the first symptoms had appeared. There had been no long remission of symptoms during this interval. The tendency toward diarrhea and vague lower abdominal pain recurred rather frequently. About two months previously the symptoms became aggravated. She began to have attacks of more severe pain around the umbilicus and in the lower right quadrant, associated with increased peristalsis and at times vomiting. The attacks suggested partial bowel obstruction. About the same time an evening rise of temperature as high as 101° F. developed and persisted with some intermissions until admission. Night sweats became a frequent complaint.

Physical examination revealed an emaciated red-headed Jewish female of normal habitus. Her weight was 114 pounds, a loss of 46 pounds since the onset of her illness. The only findings of interest were in the abdomen. It was scaphoid and soft except for muscle guarding and tenderness in the lower right quadrant, overlying a sausage-shaped tender tumor in the region of the cecum which extended over toward the midline about two inches below the umbilicus. The tumor was freely movable. The blood count revealed 5,310,000 erythrocytes, 10.5 Gm. of hemoglobin, 3,300 leukocytes, 68 per cent netrophils, 29 per cent lymphocytes, two per cent basophils and one per cent

eosinophils. Blood chemistry: sugar 111 mg., urea 10 mg., whole blood chlorides 568 mg. The urine was repeatedly normal and negative for tubercle bacilli. Six sputum specimens and several stools were likewise negative for acid-fast bacilli. The blood serologic examinations and Frei tests were negative. Some stools were negative and others positive for occult blood. No ova or parasites could be found. Sigmoidoscopy showed a normal mucosa. The secretory and motor functions of the stomach were within normal limits as determined by fractional gastric analysis.

Roentgenologic examination revealed a typical string sign in the terminal eight inches of the ileum both after the mouth meal and following a barium enema. Re-

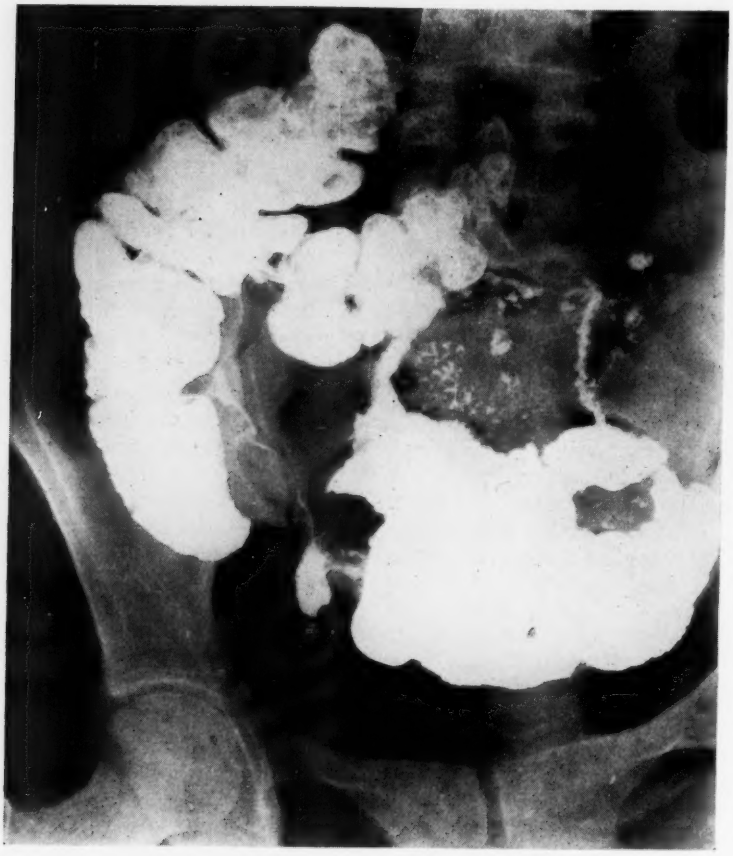

FIG. 1.—Roentgenogram showing (1) the string sign of Kantor in the terminal ileum; (2) dilatation of the terminal loop of the ileum proximal to the obstructing lesion; (3) concave shallow substraction defect on the mesial border of the cecum proximal to the ileocecal valve.

peated examinations failed to show normal filling of this segment. The cecum was not irritable but showed a slightly concave shallow subtraction defect on its mesial border just proximal to the ileocecal valve. A diagnosis of regional ileitis involving the terminal ileum was based upon: (1) a long history of intermittent attacks of pain in the lower right abdominal quadrant with mucous diarrhea but without tenesmus; (2) tenderness and muscle guarding in the lower right quadrant associated with a movable, palpable mass corresponding to the position of the terminal ileum; (3) a persistent string sign in the terminal ileum in the roentgenographic film; (4) absence of any clinical or roentgen

ray evidence of tuberculosis and the absence of signs of tuberculosis or any other type of intrinsic lesion in the cecum.

First Operation.—September 24, 1934. Laparotomy was performed by one of us through a low right rectus incision. It was found that the terminal ileum and ileocecal valve were fixed and could not be delivered. About 16 to 17 centimeters of the terminal ileum were covered by a fibrostenotic exudate and the normal glistening sheen of the wall was absent. The walls were greatly thickened and rigid, feeling very similar to a garden hose. Proximal to this region for a distance of seven centimeters there was a more acute reaction in the serosa, the vessels being engorged and the fibrostenotic exudate merging gradually into a plastic exudate. Still farther on, beginning at a distance of 24 centimeters from the ileocecal junction, the diseased segment quite abruptly merged into normal appearing bowel. The mesentery of the distal 24 centimeters of the ileum was thickened and boggy and there were several enlarged lymph nodes in the base of the mesentery near the ileocecal junction.

The presenting surface of the cecum and ascending colon did not appear or feel to be involved, but at the appendiceal region the cecum was tightly fused to the ileum by a well organized fibrotic membrane. The appendix was not visible nor could it be palpated in spite of a careful search. There was no evidence of fistula formation. While

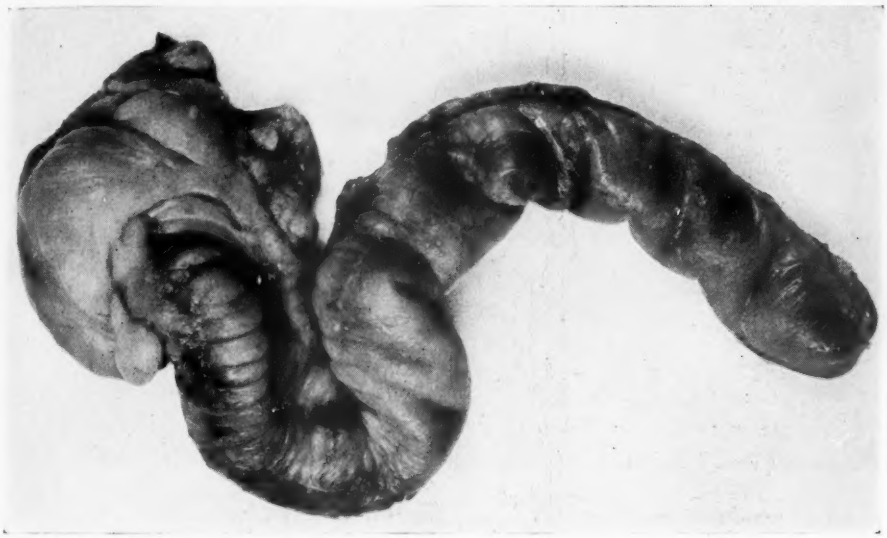

FIG. 2.—Resected specimen consisting of terminal ileum, appendix, cecum and one-half of the ascending colon.

manipulating the cecum about two to three cubic centimeters of cream-colored pus escaped from the base of the mesentery where it was adherent to the cecum, a culture of which was reported as overgrown with *B. proteus.* Because of the free pus it was deemed unwise to do a short-circuting anastomosis and resection in one stage. Consequently a side-to-side anastomosis was performed between the ileum, 50 centimeters proximal to the ileocecal valve, and the midportion of the transverse colon. The abdomen was closed without drainage. The convalescence was uneventful.

Second Operation.—After an interval of 16 days the second stage was performed. There was no important change in the gross pathology noted other than the presence of a few fine fibrinous adhesions at the site of our previous operation. At this stage the ileum was transected about eight centimeters distal to the anastomosis and the open ends were inverted. The ascending colon, which was unusually short, was transected

about at its midpoint and the open ends also inverted. The ileum, cecum and ascending colon between these transected areas were resected *in toto*. The abdomen was closed without drainage.

Following this the patient had an uneventful convalescence and was discharged from the hospital, November 11, 1934, one month after her second operation. At this time her weight was 104 pounds, ten pounds less than the weight on admission. The erythrocyte count was 5,210,000, and the hemoglobin 67 per cent (11.5 Gm.) She was having three to four bowel movements daily, normal in color and semiformed. She felt quite strong, vigorous and happy. When last seen on January 16, 1935, she had gained 34 pounds, having two to three bowel movements daily, was carrying on her housework in a normal fashion and had no complaints and no restrictions in her diet. A follow up roentgen ray examination with a barium enema showed that the ileocolostomy was functioning properly.

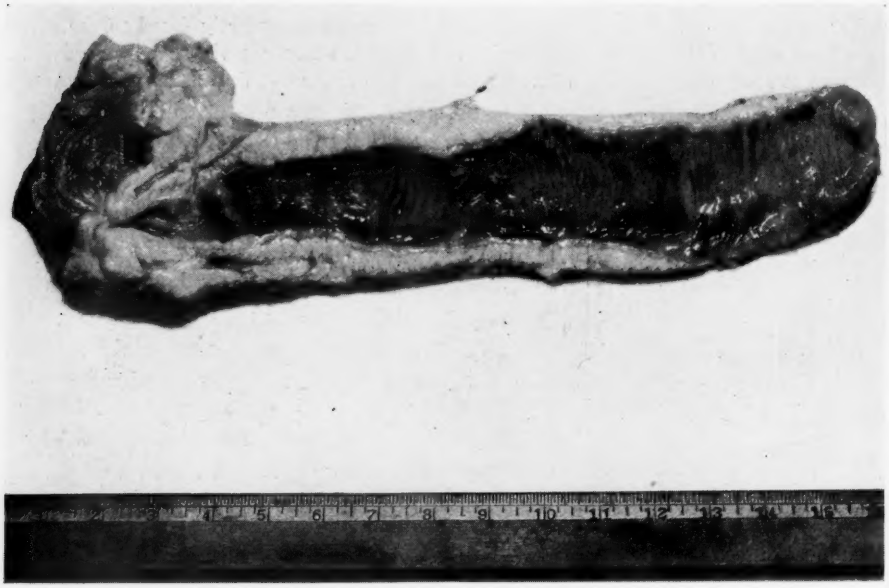

FIG. 3.—Showing the lumen of the terminal ileum, ileocecal valve, cecum and ascending colon opened. Gradual thickening of the wall of the terminal ileum toward the ileocecal valve where the lumen is almost completely obstructed.

Pathology.—The gross external pathology was as described above. Dr. Case found the appendix even on close examination remained concealed. It was entirely covered by a thick, well-organized fibrotic membrane which bound the ileum and the cecum together. The appendix under this membrane was doubled back on itself like a hairpin, and the terminal two centimeters were twisted into a corkscrew spiral. The bent portion of the appendix at its midpoint was fused tightly to the wall of the ileum about four and a half centimeters from the ileocecal valve on the mesenteric border. At this point an ulcer had completely perforated the wall of the ileum and fistula formation was prevented by the appendix becoming adherent here.

Upon opening the terminal ileum it was found that the walls reached a maximal thickness of six Mm. just oral to Bauhin's valve and tapered off as they progressed proximally until at a distance of 24 centimeters they were of normal thickness and consistency. The lumen in the distal 17 centimeters would barely admit a No. 10 French catheter. The ileocecal valve itself formed a dense fibrous ring with a three Mm.

opening in the center. The mucosa for a distance of six centimeters proximal to the valve was almost entirely replaced by dense, white, contracted cicatricial tissue, there being only a few small irregularly placed islands of mucosa present. An old scarred perforating ulcer about two centimeters in diameter was present as mentioned above, four and a half centimeters distal to the ileocecal valve on the mesenteric border. At a distance of 19 centimeters from the valve near the mesenteric groove was a small shallow ulcer one half centimeter in diameter, while just proximal to this was a larger oval ulcer measuring two and a half centimeters in its long axis, which was in the direction of the fecal flow. The mucosa elsewhere was edematous and bulbous in appearance.

The cecum and ascending colon were not involved in the pathologic process that ended abruptly at the oral side of Bauhin's valve. There were no ulcers in the mucosa of the appendix but its walls were thickened and fibrotic.

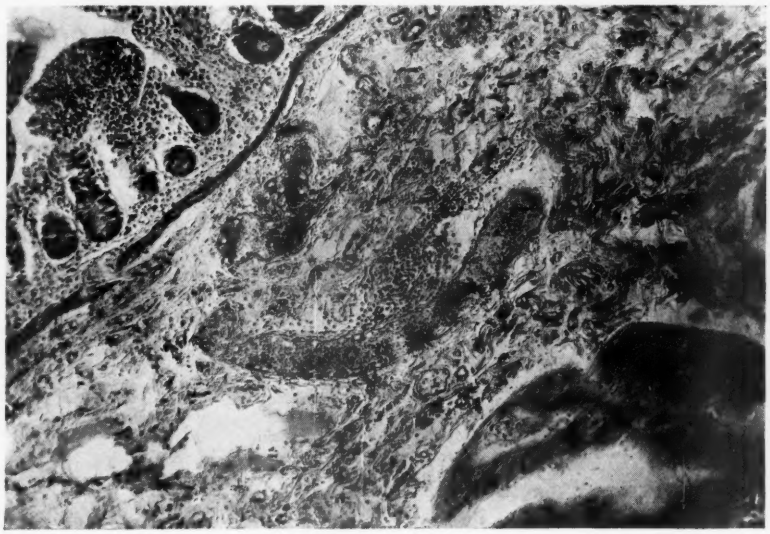

FIG. 4.—Section of the mucosa, submucosa and a portion of the muscularis. Showing a non-specific inflammatory reaction.

Microscopic Pathology.—Histologic sections revealed a non-specific, infectious, granulomatous process throughout. The chronicity of the process varied from old to recent as it progressed from the distal to the proximal regions of the lesion, respectively.

A section through an ulcer 17 centimeters from the ileocecal valve showed a complete destruction of the mucosa in the central aspect. The base was covered with fibrin infiltrated with polymorphonuclear leukocytes and the edges were undermined. The submucosa was slightly encroached upon by the ulcer, but that which remained was a dense mass of connective tissue infiltrated with leukocytes, polymorphonuclear cells being predominant. There was a slight hypertrophy of the muscular layers with evidence of the inflammatory process extending along the septums. The serosa was thickened and presented a picture very similar to that of the submucosa except that edema was a prominent feature here in addition to the inflammatory reaction.

Sections distal to the one described above varied only in the thickness of the wall of the ileum and that the polymorphonuclear leukocytes relinquished their predominance in favor of round cells and plasma cells. The walls became progressively thicker almost by mathematical precision from the proximal to the distal aspect of the lesion. There were no foreign body giant cells or tubercle-like lesions in any of the sections.

One of the neighboring lymph nodes was sectioned. Its architecture was fairly well preserved, but there was a hyperplasia of the reticulo-endothelial cells. The germinal centers likewise shared in the hyperplasia with compression and narrowing of the surrounding rim of lymphocytes. The sinuses, both central and peripheral, were widely dilated and filled with endothelial cells. There were no giant cells or tubercles present here either.

The appendix revealed a marked thickening and fibrosis of the submucus layer infiltrated largely with lymphoid and plasma cells. The muscular layers were atrophied. The subserosa was also fibrosed and showed collections of lymphoid cells perivascular in distribution. Continuous with the serosa was an organized membrane of dense connective tissue. During the process of sectioning and fixing the mucosa was destroyed and thus was not examined microscopically. However, on gross examination when the appendix was opened there was no interruption in the continuity of the mucous layer.

This case corresponds to the description of regional ileitis given by Crohn. All of the cardinal clinical features were present: youth, weight loss, fever, diarrhea without tenesmus, pain in the region of the umbilicus and right iliac fossa with vomiting, history of 14 months' duration of a palpable mass in the lower right quadrant with tenderness, and the absence of roentgen or sigmoidoscopic evidence of intrinsic disease in the colon. The cardinal roentgen finding, described by Kantor, the string sign, was well demonstrated and the evidence of intrinsic involvement of the mesial aspect of the cecum was likewise present. Grossly, this resected specimen corresponded exactly to the original description of regional ileitis. A perforation in the mesentery of the ileum had occurred and except for a barrier of the appendix, undoubtedly would have caused a fistula into the cecum. The disease process was most advanced at the ileocecal valve and just proximal to it.

Crohn, Ginsburg and Oppenheimer[1] are to be commended for the first clear-cut description of a clinical entity which in the past has probably been mistaken for tuberculosis or inoperable malignancy. Their paper is of particular importance at this time because it has stimulated interest in one of the most puzzling and difficult fields of abdominal disease, the diagnosis of small intestinal lesions. It now seems certain that the terminal ileum is occasionally the site of a progressive type of benign inflammatory lesion in which no proof of tuberculosis can be found. Speculation will continue concerning its etiology until a specific agent is found in the lesion or until a considerable number of early inflammatory lesions of the terminal ileum have been observed to terminate in what we now understand as terminal ileitis. It is not unreasonable to postulate that any primary inflammatory mucosal disease in the terminal ileum might in the end resemble the entity under discussion. There are many who feel that chronic ulcerative colitis may be originally initiated by a number of different primary agents. Certainly both amebic colitis and bacillary dysentery may eventually cause the same terminal pathologic lesion as the ordinary chronic type of ulcerative colitis of unknown etiology, possibly due to the inroads of secondary invading organisms. Hence it is conceivable that chronic ileitis may result from a number of heterogeneous primary irritating factors.

In the absence of a known etiologic agent, one should consider other reasons for the predilection of this type of lesion for the terminal ileum. It is possible that some peculiarity of the anatomy of the terminal ileum, its mesentery or its blood supply may predispose toward the development of a chronic granulomatous inflammation in this region. An arrangement which would permit of twisting, angulation, and partial intermittent intussusception or volvulus has been considered. Ginzburg and Oppenheimer,[8] in discussing the etiology of non-specific granulomas of the intestines, mention the possible rôle played by a disturbance in the vascular mechanism. They referred particularly to repeated and self-reducing intussusception or recurrent partial volvulus at the ileocecal valve, and emphasize the inability of differentiating, in the end stages, granulomatous lesions due to primary vascular insufficiency from those due to primary infectious agents. The abrupt cessation of the inflammatory process at the ileocecal valve could be explained on the basis of recurring self-reducing intussusception. The youth of most of these patients, and the lack of evidence of a specific etiologic agent, and the frequency of the involvement of the terminal ileum as well as the occasional involvement of other portions of the ileum suggested this possibility.

Cases reported to date give no idea of the anatomic configuration of the colon or the habitus of the owner of the disease. Is the disease more prone to occur in patients with a high cecum and a tendency to ileal stasis or in those with long pendulous ceca and low loose mesenteries, or does it occur without relation to these anatomic variations? According to Batson, the ascending branch of the ileocolic artery, which supplies the cecum, is more or less fixed in most anatomic specimens, whereas the terminal branch of the same artery, which supplies the terminal ileal segment, is capable of considerable rotation. It is conceivable that an unusual mobility of the terminal ileal segment might by pinching or twisting the terminal branch of this artery tend to devitalize the part in much the same way as an intussusception.

Since the disease seems to be most advanced at the valve, some primary neuromuscular abnormality here might act as a predisposing factor, inducing ileal stasis, infection, infiltration and finally obstruction.

Another possibility is primary appendiceal disease. This must be considered because of the youth of the reported patients and the history of previous appendectomy in many of them. It would not be possible in our case, for example, to exclude the possibility of a primary appendicitis with adhesion to and secondary involvement of the mesentery itself or of nodes in the substance of the mesentery. It is conceivable that a lymphangitis and surrounding inflammation might encroach upon or infect the blood supply and in this way slowly devitalize this segment of ileum, producing a lesion like that of terminal ileitis. In this classic case of ours, the appendix formed the wall of an abscess in the mesentery. The appendiceal inflammation was probably secondary, but we do not see how one can be sure of this. The

original description of the disease dismisses the appendix as a primary focus on the basis that the disease never transcends the valve of Bauhin. We feel that there is insufficient evidence so far to justify one in excluding primary appendiceal disease with secondary involvement of the mesentery and finally of the terminal ileum, in some cases.

The differential diagnosis in our case entailed primarily ruling out hyperplastic tuberculosis. The absence of lung tuberculosis and of intrinsic cecal disease tended to exclude this possibility. We are, however, very definitely of the opinion that tuberculosis should not be excluded in any case until after a microscopic examination. There is apparently no known pathologic analogy in man or the lower animals for considering this entity tuberculosis in the absence of the commonly accepted bacteriologic and tissue reactions with which all well-trained pathologists are familiar.

It is possible that the pathologic lesion under discussion may follow an acute terminal ileitis, an inflammatory reaction quite like acute typhilitis. This seems borne out by cases of the type described by De Courcy,[4]—an acute illness in a girl 20 years of age, resembling acute appendicitis following a cold in which the lower 12 inches of ileum were acutely inflamed with a plastic exudate. Clute[5] mentioned a case of this general type and Crohn discusses similar cases. It will be impossible to determine the importance of this factor until known acute inflammatory lesions of the terminal ileum have been followed over a period of years and many more sections of bowels in various stages of inflammation are resected and examined.

We feel that an analysis of the cases so far reported justifies the acceptance of the term regional (terminal) ileitis as a pathologic and clinical entity, in the same sense that chronic ulcerative colitis is used to describe a clinical and pathologic entity, recognizing the possibility of its protean etiology. Naturally, some lesions of this type will occur in combination with similar involvement elsewhere (Brown, Bargen and Weber[7]) (Homans and Haas[6]) in the bowel. Pathologic processes of this general nature have been reported in other parts of the small and large bowel. These reports in no sense detract from the clinical importance of the lesion under consideration. Rather they serve to emphasize that chronic stenosing granulomata, just as ulcerative tuberculosis, may occur anywhere in the intestinal tract, but for some reason or reasons show a marked predilection for the terminal ileum.

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RADIUM BURNS OF THE RECTUM

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CHICAGO, ILL.

SEVERE radium burns of the rectum are usually, if not always, the result of the local application or interstitial use of radium as contrasted to the effects obtained by radium packs where the element is applied at a distance from the rectum either over the sacrum or on the abdomen. Where patients with third-degree radium burns of the rectum have come under our care their burns have been either the result of treatment of carcinoma of the rectum, hypertrophy of the prostate or leukoplakia of the rectum by the introduction of radium into the rectum.

The effect of radiation on both normal and neoplastic tissues has been described by many workers in that field, and though the histologic and gross evidence of tissue changes are fairly well known, "the actual intricate mechanism by which radiation produces injury to cells is vague, and its elucidation must await prolonged biophysical analysis."[1] It is generally recognized that radiation injures the nuclei of tissue and tumor cells, causing degenerative changes in them, and also may injure the cytoplasm of the cell. The blood vessels in the irradiated area are particularly effected, hyperemia developing, followed by marked degenerative changes in the vessels, thickening of their walls and extensive thrombosis. The connective tissue is markedly stimulated in its growth and a lymphatic infiltration occurs. These are but the high lights of the reaction of tissue to radiation.

Two gross types of reaction to radium radiation are seen in the rectum; namely, hyperemia and actual necrosis or ulcer formation. Hyperemia of the rectal mucosa is practically always observed after any considerable radiation of the rectum and may be very acute, if radium, without proper screening, has been inserted into the rectum for treatment of a local lesion. It usually develops ten days to two weeks after the use of the element and increases in intensity up to three to six weeks, when it slowly recedes. The patient with such a reaction develops smarting and burning in the rectum, associated with tenesmus, which may be constant and requires morphine to control. An excess of mucus is formed which may be passed in small amounts, and if straining is severe, it may be blood stained. The rectal mucosa is plum colored, often covered with petechial hemorrhages and is extremely sensitive to touch. Such a severe reaction may cause the patient symptoms for six to eight weeks. Milder or very transitory states of hyperemia with correspondingly milder symptoms also occur.

It is, however, upon the ulceration or third degree burns following radiation that particular emphasis should be placed. These lesions may be diffuse, if proper screening of the radium was omitted, or local, in which event a

persistent indolent ulcer replaces the neoplastic ulcer that was radiated. As the hyperemia around the ulcer recedes, it has a rather characteristic appearance, being definitely outlined, rather shallow, and is covered with an exudate of fibrin and cellular débris that gives it the appearance of a white pellicle or membrane. Due to connective tissue contraction, radiating lines of constriction are seen in the mucous membrane surrounding the ulcer and the mucosa appears smooth and atrophic, without its usual delicate folds. In four of our patients the ulcer was over the prostate at the former site of the carcinoma, and was firmly adherent to the prostate and immobile. In one patient a small lesion the size of a dime had replaced the site of an adenoma destruens. In two patients the ulceration was diffuse, as a result of treatment of hypertrophy of the prostate and leukoplakia respectively.

Histologically, the picture was typical of ulcers following radiation in any part of the body. A dense, unorganized necrotic base, under which marked fibrous and leukocytic infiltration was present. Damaged nuclei in the cells were in evidence. The walls of the blood vessels were thickened. Practically no evidence of healing was in evidence except perhaps the contraction of the ulcer by fibrosis. For purposes of discussion eight cases with marked radium burns of the rectum are selected.

In six of them the burns resulted from treatment of carcinoma of the rectum and in two from treatment of hypertrophy of the prostate and leukoplakia of the rectum respectively by the local application of radium.

Of the six radium burns occurring following treatment of carcinoma there was suspicious gross evidence of carcinoma in two (Figs. 1 and 3). In the center of these ulcers there were some irregular nodules much firmer and less friable than usually felt in adenocarcinoma of the rectum, but nevertheless highly suspicious. In the other four patients the lesion was a flat ulcer with marked scar tissue contraction about it and had no characteristics of a carcinomatous ulcer. When these six radium burn ulcers were examined histologically, three had very definite evidence of carcinoma in the depth of the ulcer under the necrotic exudate (Figs. 2 and 4), and in two degenerated cells were found in the base of the ulcer which were histologically very suggestive of carcinoma, though that evidence must be somewhat less definite than usual due to the badly damaged condition of the nuclei of all the cells in that region (Fig. 5). In the sixth patient, aged 79, where an adenoma destruens had been removed and radium needles planted into the rectum at its base, the burn resulting was not in any way typical of carcinoma and repeated biopsies over a period of five years showed no tumor cells. This patient died of pneumonia and no postmortem was obtained.

In the five patients having radical removal of the rectum a careful dissection of the specimens for involved lymph nodes showed positive findings in one instance. This patient was a young man 30 years of age in whom early extension into the lymphatics would usually be expected. As the remaining cases were from 30 to 62 years of age and had had symptoms of carcinoma before the radium treatment for from four to six months (with one excep-

tion), it is not worthy of particular comment that only one had glandular involvement, as the average patient has only 35 per cent of a chance of having such involvement when operated upon.

Pathologically considered, it seems reasonable to say that in these six patients with radium burns following the treatment of carcinoma of the rectum by radiation, three showed definite evidence of carcinoma, two probable evidence and one, no evidence of tumor cells in the burned area.

Clinically speaking, Case VI, with the small burn following radiation of the adenoma destruens, was only moderately uncomfortable, having a slight bloody discharge and some slight pain but in general was in good health. The remaining seven patients, including one with an extensive burn following radiation of an hypertrophied prostate, were practically incapacitated. Marked tenesmus, frequent evacuations of blood, mucus, bladder irritability, sleeplessness, loss of appetite and anemia were present in varying degrees.

From a surgical standpoint the radical removal of the rectum was made very difficult because of loss of the normal cleavage planes by reason of extensive fibrosis and also because of dense adherence of the radium burn to the prostate in three patients, in whom it was necessary to remove the posterior one-third to one-fifth of the prostate by knife excision to entirely remove the necrotic area of the burn. In these patients the prostate looked edematous and fibrotic, as if it had also been markedly affected by the radiation. Following operation a white exudate formed over the prostatic bed which persisted for weeks but finally cleared up. Persistent oozing at the operation necessitated transfusion of blood in nearly all of the cases. The postoperative course was stormy in all, as pain, slow healing of the wounds and profuse wound discharge complicated the convalescence. In the patient with extensive burn following treatment of prostatic hypertrophy, the rectum was split open posteriorly after removal of the coccyx and the burn was thoroughly excised and the adjacent mucosa mobilized to cover the defect as much as possible. All of the patients recovered from the operation. One has since died of carcinoma, having local as well as general recurrences. The remaining six are well and free from recurrence, one to two and one-half years following the operation.

In conclusion, it seems evident from this small series of cases that when carcinoma of the rectum is treated by radium and a burn results, that carcinoma may still be present in the base of the burn or in the lymphatic nodes and that radical removal of the rectum is indicated. If radium burn in the rectum results from treatment of other lesions, local removal of the burn is the treatment of choice as applies in radiation burns elsewhere in the body.

Case Reports

Case I.—I. K., male, aged 56. Diagnosis of carcinoma of rectum five months ago. Ulcer over prostate, treated by insertion of radium into the rectum at two sittings, 2,800 mg. hours administered. Following treatment symptoms improved but now he has bleeding and tenesmus and has lost weight. Examination May 12, 1932, shows scar tissue contraction at level of prostate with some induration and superficial ulceration.

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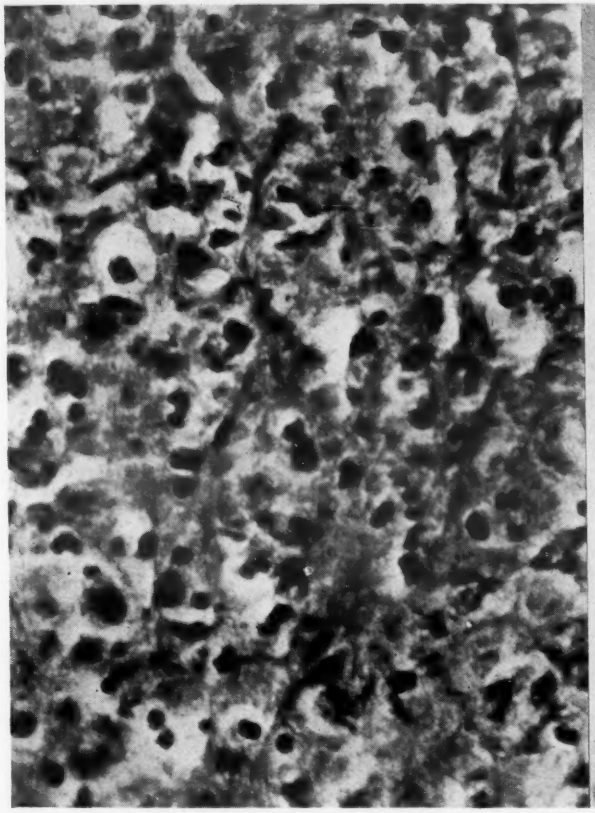

FIG. 1.—(Case II).—High power photomicrograph of tissue in depth of radium burn of rectum where tumor has grossly been destroyed. Questionable tumor cells.

FIG. 2.—(Case III).—Gross specimen of resected rectum showing radium burn with tumor tissue in it two years after local application of radium, dosage unknown. See Fig. 3 for histology.

Operation May 21, 1932, exploratory laparotomy and colostomy. May 4, 1932, posterior resection of rectum. Four inches of bowel removed intraperitoneally. Much oozing. Transfusion of 500 cc. blood. Specimen shows ulcer two and one-half inches by one inch on anterior wall. Adherent to prostatic tissue. Sections show degenerated cells resembling carcinoma in base of burn. Lymph nodes negative. Stormy convalescence. Prostatic area has all appearance of radium burn but eventually healed. Patient now well and at work.

CASE II.—C. F. M., male, aged 50. Symptoms of blood and increase in number of stools for three months. Examination February 15, 1932, shows carcinoma size of dollar on posterior wall of rectum with crater in center above mucocutaneous line. Lesion is freely movable but involves muscularis.

Radium treatments were administered elsewhere in this case in March, 1932. The first consisted of 48 mos. filtered with 1 Mm. brass and ½ Mm. silver placed high up in the rectum against the growth for eight and one-half hours. Two days later, three tubes of radon

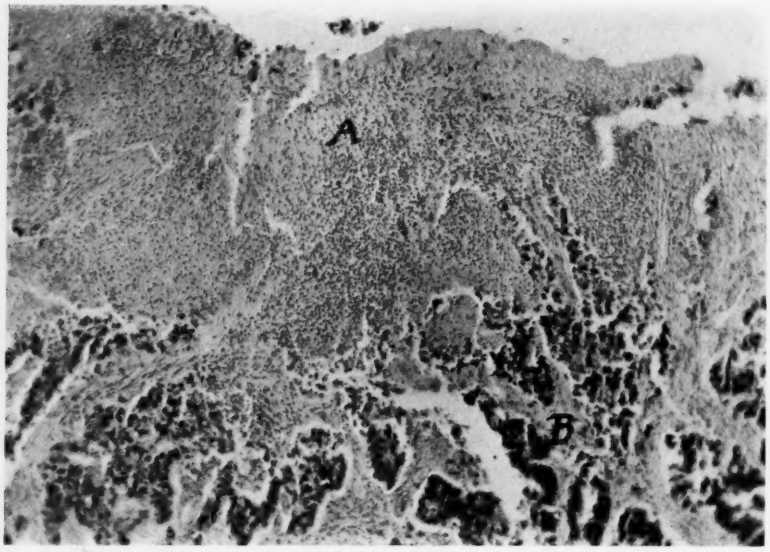

FIG. 3.—(Case III).—Photomicrograph showing necrosis of radium burn (A) and tumor cells in the depth of the burn (B).

totalling 129 mos. were placed for four hours against the lesion, held in place with gauze packing. Two days later a similar application was placed lower down on the lesion, and two days later a similar treatment was given. The total of these four treatments between March 8 and March 14 was 1,973 mo. hrs. On March 17, 17 mg. platinum needles were placed throughout the rectal growth one cm. apart, and left in place for 48 hours. A very severe reaction followed, including a pulmonary embolism. Ever since has had tenesmus, 6 to 50 bowel movements daily.

On June 21, 1932, was bedridden, taking morphine, in continuous pain and was pale and emaciated. Digital examination of rectum shows no tumor, marked scar tissue formation and superficial ulceration at its former site. June 23, exploratory laparotomy and colostomy. Liver and lymph nodes negative. July 22 blood transfusion 500 cc. Posterior excision of rectum. Eight inches of bowel removed. Prostatic bed edematous. Marked fibrosis in region of middle hemorrhoidal vessels. Ulcer from radiation two and one-half by two inches on lateral wall of bowel. No gross evidence of carcinoma. Microscopically, very probable carcinoma cells in depth of ulcer (Fig. 1). No involvement of lymph nodes found. Patient made a slow convalescence. At present, the patient is well except for a phlebitis. No evidence of recurrence.

CASE III.—S. W., male, aged 62. In June, 1931, carcinoma of rectum diagnosed elsewhere and treated by radium. Dose unknown but given by application in rectum. In good health until present, November 20, 1933, when blood in bowel movements recurred. Examination shows flat ulcer about two and one-half inches in diameter at level of prostate and adherent to it. Dense scar tissue about it. Operation November 29, 1933. One stage abdominoperineal resection followed by blood transfusion. Posterior one-fifth of prostate was removed with the adherent ulcer. Flat ulcer in rectum with definite carcinoma in center of it (Fig. 2). Microscopically cancer (Fig. 3). Lymph nodes negative. Patient made excellent recovery and is now in good health.

FIG. 4.—(Case IV).—Gross appearance of radium burn of rectum following use of 146,000 Mg. hours by radium packs and 3,000 Mg. hours by rectal applicator. Note questionable tumor nodules and flattening of normal mucosal rugae. For histological picture see Fig. 5.

FIG. 5.—(Case IV).—Photomicrograph showing tumor cells in depth of burn.

CASE IV.—J. J. S., male, aged 61. Five months ago frequent bowel movements containing old blood. Lost 30 pounds in weight. Examination May 19, 1933, shows a movable carcinoma two and one-half inches in diameter just above the mucocutaneous line on the left side, reaching from prostatic region to hollow of sacrum. Biopsy showed carcinoma. Patient was treated elsewhere with radium. 146,000 mg. hours with the four gram radium pack, 10 cm. distance, 10 cm. portal, 1 Mm. platinum filtration was administered in two series: July 11 to 28, 1933 and October 3 to 13, 1933. In addition to the external radiation he also received radiation by means of a rectal applicator as follows: 30 mg. for 100 hrs., 1 Mm. platinum, total dose 3,000 mg. hours.

Patient returned November 16, 1933, suffering severe pain. Had lost more weight. Examination shows marked scar tissue contraction but very little, if any, evidence of tumor. Large ulcer adherent to prostate and immovable. Two blood transfusions were given and the rectum removed abdominoperineally in one stage (Figs. 4 and 5). It was necessary to remove the posterior one-fourth of the prostate with the adherent ulcer. Stormy convalescence with eventual recovery and subsequent return to work.

CASE V.—F. D., male, aged 31. In February, 1932, polyp removed from rectum elsewhere which was subsequently diagnosed carcinoma. An experienced radiologist treated the area with 100 mg. of radium contained in two 50-mg. tubes in tandem, applied for a period of 15 hours. Screened by five Mm. of platinum, one Mm. of lead and one Mm. of aluminum. The distance from the radium to the mucosa was a minimum of 2.5 Mm. Following this, patient had a severe local reaction and eventual ulceration, followed by stricture, great difficulty in having bowel movements which contain blood and pus. May 7, 1932, there was an ulcerating lesion on the posterior wall of the rectum involving the sphincters and a stricture at the level of the levator muscle. A deep biopsy of the ulcer was made but no carcinoma was found. A colostomy was done, hoping to give the patient relief from severe pain. June 8, 1932, severe pain continuing it was decided to excise the radium burn. While so doing a nodule in the base of the burn was seen which proved to be carcinoma. A radical excision of the rectum was then carried out from below, opening the peritoneum and removing about eight inches of the rectum. Other carcinomatous nodules were found along the superior hemorrhoidal vessels. Examination of the base of the radium burn showed carcinoma. The patient made a good recovery and returned to work, but on November 24, 1933, returned with sciatica and eventually developed lung metastases, as well as a local recurrence, and died September, 1934.

CASE VI.—L. G. Y., male, aged 76. April 11, 1924, blood in stool. Above prostate on anterior wall is a pedunculated movable polyp with indurated base. Feels like adenoma destruens. April 22, 1924, removed by cautery, after splitting rectum open posteriorly. Four needles containing ten mg. each implanted into previous site of tumor and left five hours. November 28, 1924, slight ulceration at previous site of tumor, white and covered with a pellicle. Biopsy shows necrotic tissue but no carcinoma. November 26, 1926, same picture and again biopsy negative. July, 1927, picture the same, patient having no symptoms. In 1929, died of pneumonia. This lesion was apparently cured by local removal followed by radium.

CASE VII.—N., male, 63. Symptoms of large prostate treated elsewhere by having radium inserted into rectum two months ago. Now having severe tenesmus. Passage of bloody mucus, is sleepless and losing weight. Examination shows area three inches in diameter on anterior wall of rectum, ulcerated and covered with a white exudate. The surrounding mucosa is deeply injected and bleeds easily. No suspicion of carcinoma. The rectum was split open posteriorly, the area of ulceration excised, the mucosa adjacent to it mobilized and the defect partially covered by it. The rectum was sutured in layers. Immediate relief from pain and favorable convalescence. Histologically the tissue showed a radiation burn with no evidence of carcinoma.

CASE VIII.—F. M., male, aged 30, has marked leukoplakia of the rectum, with thick parchment-like rough mucosa, extending upward for about two inches above the mucocutaneous line. This was excised and promptly recurred. Following recurrence patient went elsewhere and had radium introduced into the rectum. A few weeks later he developed severe pain and on returning for examination was found to have a disappearance of the leukoplakia, but a radium burn involving an area about one and one-half inches long by three-quarters of an inch wide. This was excised and the normal mucosa pulled down over the area. The amount of radium used in this case was not known. Following this operation the patient made a good recovery.

REFERENCE

[1] Stewart: Arch. Surg., vol. 27, p. 97-A, 1933.

THERMAL BURNS

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THERE has been in the past decade such a wealth of literature, both clinical and experimental, on the subject of burns that it is difficult to find a reasonable excuse to increase it. Yet as the interested physician reads the various clinical opinions and studies experimental results by competent observers, he is immediately conscious of a lack of harmony in not only the theoretical and experimental results, but also in the methods of treatment. He is also conscious of improved mortality statistics but the improvement seems disproportionately small when compared with the advancement in knowledge claimed. He is confronted with experimental data purporting to show that the fatal effects of burns are due variously to a reflex phenomenon, increased capillary permeability, a tissue toxin or a bacterial toxin. He is amazed that the proof in each case seems conclusive. The statistical studies from the various clinics all show improvement but in each instance there are essential differences in methods of treatment and each clinic is convinced that its procedure is correct.

In this paper an attempt will be made to harmonize both clinical and research data and to outline a method of treatment based upon the conception that the phenomena induced by thermal burns are in reality a sequence of events and should be treated as such. This seemingly impossible task simplifies itself immediately when one separates from the mass of clinical and experimental evidence certain basic facts, all of which have been reasonably proven. First, the phenomena induced by burns are not only local but also general in action. Secondly, there are definitely reflex, biochemical and toxic phenomena. An effort will be made to show that each of these facts is probably true, to place them in their proper order, and to evolve a sequence of therapy which will not only actively combat but will anticipate and prevent their ill effects. This effort to harmonize is the only claim to originality that the author makes.

There is certainly nothing new or revolutionary in the idea that the cause of death in severe burns is due to general and not local phenomena. That the general factor is precipitated by the local lesion is obvious. As early as 1868 Wertheim[1] advocated the idea of a toxin circulating in the blood as the prime factor in burn mortality. That he was not alone in his attitude is illustrated by Pontick[2] advocating the same idea in 1876. A number of rather unique theories were put forth at this time to explain the phenomena.

The theory that the burn decreased the respiratory function of the skin to the point where life was impossible is as unique as it is untenable in the light of present-day physiology. The theory that the toxicity was due to the retention of products normally excreted by the skin was disproved by Kijanitzin[3] in 1893 when he demonstrated that the ill effects of the gilding experiments were due to abnormal biologic conditions produced in the skin and that the gilding destroyed the vitality of the skin involved, producing a microscopic picture not unlike that seen in a first-degree burn. Welti[4] in 1889 disproved the theory of death being due to failure of the heat-regulating mechanism by protecting the burned animal from such heat dissipation and showing that there was no reduction in mortality. Markusfeld and Steinhaus,[5] in a series of brilliant experiments, demonstrated that the interference with the nerve supply of the burned animal had no effect upon the ultimate result, while division of the blood supply to the burned area prevented the constitutional reaction.

That the first ill effect of a severe burn may be a reflex shock phenomenon, due to pain and fright, has been adequately proven by Underhill, Kapsinow and Fisk.[6, 7, 8, 9, 10] These investigators noted that in animals burned under anesthesia the primary shock seen in the clinical case did not occur. Furthermore, Underhill and his colleagues showed that the primary shock occurred too quickly for it to be caused by an actual reduction of the total blood volume, and that the loss of the power of absorption from the burned area in the first 12 to 24 hours following a burn precludes the explanation of a protein absorption product. That the primary shock could be due to a bacterial toxin is untenable in the light of our knowledge of bacteria and their toxins and the lack of absorptive power in the burned area in this early stage likewise precludes this possibility. From the above data one can therefore state with reasonable certainty that in severe burns there may be a primary shock due to pain and fright.

The primary shock, however, is a transitory phenomenon to which the patient either succumbs, or responds to treatment within a relatively short interval. It may be followed by a secondary stage which, in its clinical symptomatology, could be likewise called shock. This for the sake of clarity we will call secondary shock or the second stage of burns. It is concerning the etiology of the secondary shock that most of the controversy rages. It was generally accepted up to the last few years that the ill effects of burns during this stage were due to toxins liberated in the burned area by protein disintegration which were absorbed into the circulation. The leading proponent of this theory was Davidson[11] who in 1925 summarized the evidence in favor of this hypothetical toxin and presented the tannic acid treatment of burns. Although Mitchiner[12] claims the Chinese used this treatment five thousand years ago when it was their custom to treat burns with tea leaves, it does not detract from the credit which is due Davidson for instituting the first major advance in the modern treatment of severe burns.

To uphold the tissue toxin theory we have the work of Reiss[13] in 1904

who reported a toxin in the urine of the burned case which was toxic upon being injected into the laboratory animal. Pfeiffer[14] in 1905 reported the isolation of cleavage products from the burned skin which were neurotoxic and necrotoxic. Others too numerous to mention conducted investigations to show the presence of this toxin. It remained, however, for Robertson and Boyd[15] in 1923 to present the strongest evidence for this theory. Briefly summarized these investigators grafted burned skin onto normal animals who showed toxic manifestations in about eight hours. They injected blood from burned animals into normal animals and found the whole blood to be highly toxic whereas blood serum alone was nontoxic. The injection of alcoholic extracts of normal skin were found to be nontoxic to normal animals while the injection of alcoholic extracts of burned skin were immediately followed by toxic symptoms. These investigators isolated and analyzed this toxic and found it to be of two parts. The first was thermostabile, diffusible and neurotoxic, and the second, thermolabile, colloidal and necrotoxic. The toxin, chemically, consisted of primary and secondary proteoses. From these experiments the earlier idea of débridement and exsanguination transfusion evolved.

In recent years Underhill, Blalock, Wilson and their respective colleagues[6, 7, 8, 9, 10, 16, 17, 21] have attacked the tissue toxin theory vigorously. These investigators, working independently, have obtained results so convincingly harmonious that a rapidly increasing group now deny the existence of the tissue toxin entirely, and a still larger group admit that if the tissue toxin exists at all it is responsible only in part for the deleterious effects of burns. Both of these investigators point out the lack of unified opinion among the early works of Reiss,[13] Lustgarten[18] and Spietschka,[19] and that the results they obtained do not occur with convincing regularity. Furthermore, very few of these investigators have been able to check the results of the others. Underhill and Kapsinow[16] repeated the experiments of Robertson and Boyd exactly. They were unable to demonstrate the circulatory toxin in that the injection of whole blood from burned patients did not produce toxic manifestations in normal animals. They did, however, find the injection of an alcoholic extract of burned tissue to be toxic to normal animals but they likewise found that the injection of an alcoholic extract of normal skin was also toxic. An analysis of the extract showed enough alcohol to be present to account for the symptoms observed in the opinion of these investigators. They consequently injected into normal animals an equal quantity of ethyl alcohol and produced identical symptoms. They, therefore, concluded that the primary and secondary proteoses of Robertson and Boyd were, in reality, ethyl alcohol.

Harrison and Blalock[17] grafted burned skin onto normal animals and stated that they could find no evidence of toxemia. These investigators also burned animals and found that the shortest survivals were among those débrided. They also confirmed Underhill and Kapsinow's results with whole blood injections from burned animals in that they could demonstrate no toxin.

The most convincing evidence against the tissue toxin theory, however, are the experiments of Underhill and his colleagues with burned tissue showing that, whereas burns induced an increased capillary permeability, the loss of fluid is external and that the absorption from the burned area does not take place at all for 12 hours and but very slowly for 24 hours. In these experiments the injection of methylene blue into the burned area showed no absorption into the blood stream and, more convincing, strychnine injected in lethal doses did not even produce convulsions. Certainly this is proof enough that if tissue toxin exists at all it cannot be a factor in the secondary shock stage.

Fortunately, in attacking the tissue toxin theory Underhill placed before the profession another theory to account for the secondary shock stage and produced evidence in its favor that is reasonably incontrovertible. Underhill, Carrington and Kapsinow,[20] in 1923, working on a number of burned cases, demonstrated the remarkable concentration of the blood which we now accept as an associate of severe burns. Experimentally Underhill and his associates have enlarged upon this observation until they have proved beyond reasonable doubt that the secondary shock stage is, in all probability, due to biochemical changes alone. As has been pointed out previously, a large group of clinicians while recognizing that these biochemical changes account for most of the phenomena observed, still insist that a toxic protein is absorbed also, and the two factors together account for the secondary shock.

As previously stated, Underhill and his associates claim and present experimental evidence to show that when an animal is burned there is an increase in capillary permeability in one direction and a decrease in absorptive power in the other. In other words fluid pours rapidly from the capillaries out into the intercellular spaces and onto the surface whereas no reabsorption takes place in the burned area for 12 hours and then very slowly for 24 hours. They have measured and analyzed this fluid. It is estimated by these investigators that, by comparison to the experimental animal, a man weighing 150 pounds with one sixth of his body surface burned will lose 3500 cc. of fluid in the first 24 hours and that the fluid lost is furthermore directly proportional to the surface area involved. With these figures in mind it is readily seen that our ideas of the fluid requirements of the burned patient need revising. Furthermore, analysis of the fluid shows that it is so similar, chemically, to blood serum that for all practical purposes it could be considered identical. Also, it is shown that there are no blood chemical changes in the burned animal which could not be due to this fluid loss. As additional proof the lowering of the blood pressure follows exactly the curve of the fluid loss. Blalock[21] confirms in his experiments the results of Underhill. The conclusion is drawn, therefore, that whereas the primary shock is due to a relative reduction of the total blood volume, the secondary shock is due to an actual reduction of the total blood volume.

The very latest of the theories concerning the deleterious effects of thermal burns is reported by Aldrich[22] who generously ascribes the idea and the

resulting proof to Firor. While discounting the tissue toxin theory entirely, and admitting the probability of Underhill's blood concentration theory, this investigator believes that a bacterial toxin is responsible for a good proportion of the systemic manifestations of burns. It is difficult to classify this theory in either the second or the third stage of the burn phenomena, as Aldrich claims it accounts partly for the symptomatology of the second stage and entirely for the symptomatology of the third stage. He cites in proof of the existence of this bacterial toxin in the stage of secondary shock that burned patients succumb during this period even when the concentration of the blood has been restored to within normal limits. However, as Brandson and Hillsman[23] have pointed out, the restoration of the blood to normal concentration of red cells in the second stage is usually accomplished by the injection of isotonic saline intravenously and such therapy does not take into consideration the serum protein deficit. The lowering of the serum protein content of the blood alone is sufficient to cause shock-line symptoms and death, even in the presence of a normal concentration of the blood as estimated by the red cell count and hemoglobin index.[36] However, Aldrich[22] took cultures of the burned area before beginning treatment and repeatedly at intervals afterward. For the first 12 hours the cultures were sterile but after this period had elapsed the cultures were invariably positive. In the fatal cases hemolytic streptococci were found in the heart blood and in the lungs. From this theory Aldrich evolved the gentian violet treatment which is rapidly coming into favor.

The third stage of burn symptom is generally admitted to be due to bacterial infection and (Aldrich notwithstanding), is not invariably present. This is a comparatively late stage and the complications of burns usually appear during it.

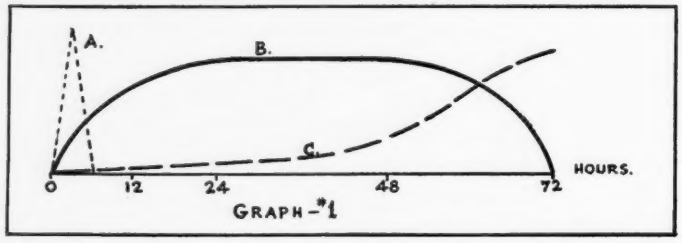
With the foregoing discussion one can readily see that the phenomena of severe burns must be visualized as a sequence of events, and that the physiologic and biochemical changes occur in an orderly manner. At the onset of a thermal burn several things occur. First there are pain and fright which may induce reflex shock, represented by a line A (Graph 1). Coincidentally an increase in capillary permeability is induced with loss of fluid. This starts immediately, reaches its peak in 24 hours, is stationary for 24 hours, and then is slowly reabsorbed. This is represented by line B (Graph 1). At the same time bacterial contamination occurs which may become active infection in 36 to 48 hours and induce toxic symptoms. This is represented by line C (Graph 1). If one believes in the tissue toxin theory its place in the sequence would be represented by B as seen in Graph 2.

If the Aldrich theory of bacterial toxicity is to be considered, then it must be combined with Underhill's blood concentration theory and would be represented by line C (Graph 3).

In summarizing the sequences, the author, while leaning strongly to the sequence as represented in Graph 1, gives all the possibilities because when

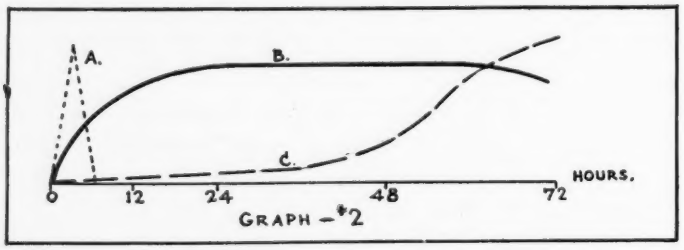
the therapeutic régime he advocates is outlined it will be seen that the possibilities of all three sequences will be taken into consideration.

PATHOLOGY.—The examination of postmortem material shows conclusively that the pathologic changes induced by severe burns are not confined



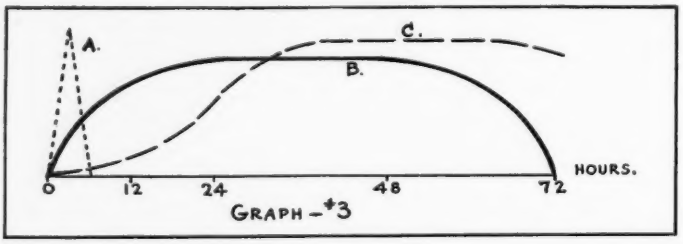
GRAPH-*1

solely to the local and obvious lesion. Depending upon the extent and severity of the burn there are very definite lesions produced in almost every organ in the body, lesions which may in the final analysis be responsible for the death of the patient. Certain of these systemic changes are relatively



GRAPH-*2

easy to explain, such as the characteristic amyloid changes which occur late in burns as the result of long continued suppuration. Other lesions, however, are more difficult to understand and numerous explanations have been offered. The more important of these theories are the reflex, the blood concentration, the tissue toxin and the bacterial toxin already discussed.



GRAPH-*3

The variations in the character of the local lesion in burns depend upon a number of factors, the more important of which are, the intensity, character, and duration of the heat. According to the local changes observed heat burns have been divided into degrees. These degrees are necessarily arbitrary and in actual clinical work not clearly defined. Some authors will describe only three degrees while others describe as many as six. For a

classic description of both the local and general pathology of burns one may refer to the monograph of Pack and Davis.[24]

First Degree.—This consists of a simple erythema with vascular reactions similar to that of any simple inflammation due to the action of an irritant. It is essentially a reflex and exudative reaction. The reflex phase consists of contraction followed by dilation of the arterioles and venules, resulting in an increased flow of blood to the affected area and a local stagnation of the current. This naturally causes the redness and increased heat invariably associated with this type of burn. The exudative reaction is explained by an increased capillary permeability and consists of filtration of plasma into the tissue spaces, the migration of leukocytes, and the diapedesis of red blood cells. Within a few days the outer layer of the epidermis separates and peels off.

Second Degree.—This is essentially the degree of vesication. The epidermis undergoes true coagulation necrosis. By virtue of the increased capillary permeability there is an exudation of serous fluid and filtration of leukocytes from the tips of the papillae, resulting in the formation of blebs or bullae. In mild cases the exudation occurs within the epidermis and leaves the basal cell layer intact, thus forming the bleb. In the more severe cases the fluid collects in larger amounts beneath the full thickness of the epidermis, resulting in the larger and more serious bullae. In the very severe cases the fluid may even resemble plasma and in such cases organization of the fibrinous exudate is more likely to follow than absorption. Such blisters should be evacuated before organization occurs. It is well to remember that in this type of burn scarring may take place, particularly if infection complicates the picture.

Third Degree.—In this the epidermis is entirely destroyed as is part of the corium. Its distinguishing factor is that the interpapillary processes remain intact. The papillae appear as a reticular framework containing serum, bits of persistent living epithelium, leukocytes and fibrin. These papillae show as red points on a white background. Two things should be noted about this type of burn. First, the exposed nerve ends in the interpapillary processes remain intact and as a consequence this is the burn accompanied by the most severe pain. Secondly, the sebaceous glands, the hair follicles and the sweat glands are intact and will regenerate new epithelium providing infection does not destroy their epithelial cell lining.

Fourth Degree.—In this there is destruction of the entire thickness of the integument and complete disorganization of the skin involved. If the traumatizing factor is dry heat this area will vary from brown to black in color, be insensitive and leathery to touch. If on the contrary moist heat is the agent the affected area will be white, insensitive and finger pressure will not induce the usual color changes. Surrounding this zone will be areas of lesser degree burns, shading off from the third degree to the simple hyperemia of the first degree. An acute inflammatory process starts very early and the eschar retracts leaving a groove between the dead and living tissues.

Sloughing of the dead tissue begins and is completed normally within two weeks. At the same time proliferation occurs. Fibrinous exudation upon the surface exerts a chemotactic action upon the new tissue cells. The capillaries proliferate tufts of endothelial cells accompanied by new growth of fibroblasts, to grow along the fibrinous framework after the manner of healing by secondary intention. The endothelial cells hollow out to form arches of new capillaries giving nourishment to the growing tissue. Scarring and contracture deformities result.

Fifh Degree.—This type of burn varies only from the fourth degree in that the underlying muscles are involved. The resulting scar is, as a consequence, more deforming and may cause great functional impairment. The scar has a great tendency to break down and ulcerate.

Sixth Degree.—In this degree the tissues are carbonized and the heat may even fracture the underlying bone. It is usually seen in the very severe type of burn involving the digits and demands, invariably, amputation of the part.

The pathology of the internal organs is not by any means characteristic in burns. It has been noted that the changes were similar to those found in the acute infectious diseases of the skin. At necropsy the following systemic changes have been noted.

Central Nervous System.—Hyperemia of the brain and meninges with small minute hemorrhages into the brain substance and engorgement and thrombosis of the veins of the arachnoid.

Kidneys.—Marked changes are found in the kidneys. Acute glomerulitis is an early finding with cloudy swelling and fatty degeneration appearing later in the proximal convoluted tubules. Thrombi have been noted in the vessels, particularly in the glomerular tufts. Necrotic areas are found in the more severe degrees of burns and hemoglobin occurs throughout the renal cortex to give the kidney the brownish-red color seen almost invariably in fatal burns.

Suprarenal Glands.—The pathologic findings in these organs are most interesting because attempts have been made to ascribe the fatal effect of burns to disturbance of their function. The normal weight of the suprarenal gland is from four to seven Gm. and in the severely burned case it often weighs from 20 to 25 Gm. Experimentally within 24 hours there is marked hyperemia and occasional hemorrhages noted in the glands. The perirenal fat is markedly edematous. Later the glandular cells are swollen, hydropic and frequently show necrosis.

Spleen.—The spleen is enlarged and shows focal necrosis in the germinal centers of the lymph nodules. The endothelial cells proliferate rapidly to occupy the germinal centers and may fuse to form giant cells. Later hyaline degeneration of the lymph nodules may be seen.

Heart.—This organ in the late severe cases shows hyaline and fatty degeneration with necrosis of the muscle fibers.

Liver.—Parenchymatous degeneration with focal necrosis and hyperemia is usually seen in this organ.

Lungs.—In fatal cases of burns the lungs are found congested with thrombi in the smaller branches of the pulmonary artery. Focal pneumonia is usually present.

Lymph Nodes.—The usual lesion seen in the lymph node is central necrosis of the follicles with endothelial proliferation.

Gastro-Intestinal Tract.—Hyperemia is noted throughout the gastro-intestinal tract with petechial hemorrhages into the mucosa. Ulcerations may occur anywhere along the tract but are found most frequently in the duodenum.

COMPLICATIONS.—In discussing the complications of thermal burns one might justly say that all complications begin with and include the third stage. Since in the preceding outline it was stated that the third stage was that of infection and (Aldrich notwithstanding), was not an invariable sequence of all burns, then the occurrence of infection itself could be classed as a complication. The very character of the trauma naturally produces a lesion which is at first sterile. However, in a burn involving any great extent of the body surface it is manifestly impossible to prevent subsequent contamination. Whether this contamination goes on to actual infection depends upon a number of well known factors. It is certain that a high percentage of severe burns do reach the actual stage of infection and a goodly proportion actively suppurate. The presence of necrotic tissue as an excellent culture medium together with inevitable bacterial contamination makes this easily understandable. To keep bacterial contamination at a minimum and to take every precaution against its occurrence constitutes one of the chief aims in the therapy of burns. It lessens the frequency of development of the following complications:

Septicemia.—This complication, while reported rarely, may well occur more frequently than is realized. Aldrich[22] found streptococci in the heart's blood of all his fatal cases of burns.

Erysipelas and Tetanus.—Both of these conditions complicate burns but fortunately occur in only a small percentage of cases.

Hemorrhage.—Hemorrhage occurs in a small percentage of cases and is usually met with only in dirty, sloughing, badly infected burns. It occurs from the superficial veins and infected granulation tissue. Since the burned patient is usually in poor condition when this complication occurs it is quite serious.

Bedsores.—Bedsores are seen in burned cases among the aged and those cases who are bedridden over long periods without proper prophylactic attention. This complication is also fortunately rare.

Pneumonia.—Pneumonia is one of the more common complications in fatal cases of burns. In those cases dying late in the course of the sequence, pneumonia or nephritis are the usual cause of death.

Nephritis.—Kidney damage occurs early in the sequence of burns. In fact, so regular and so early does this complication occur it may be taken as a part of the phenomena of burns and not classified as a complication. The

early occurrence of kidney damage warns us that unguarded intravenous fluid therapy after 48 hours may be dangerous. The early kidney damage may be accounted for by any of the numerous theories advanced but the late stage is undoubtedly due to bacterial toxicity.

Amyloid Degeneration.—Amyloid degeneration is not a peculiarity of burns but is found in this condition, as elsewhere, after long-continued suppuration.

Curling Ulcer.—Although ulcers have been reported throughout the gastro-intestinal tract following burns, the more common location is in the duodenum. This variety of ulcer is one of the more interesting and inexplicable facts we have in surgery. It was first described by James Long[25] in 1840 and later by Curling[26] in 1842. Although they are rarely reported in the literature they probably are, in reality, a frequent complication. Perry and Shaw[27] reported duodenal ulcers as being routinely found in 0.4 per cent of all cases coming to autopsy and in 3.3 per cent of all persons autopsied after severe burns. Since these ulcers do not produce symptoms unless perforation or hemorrhage occurs and since the mortality under these conditions is given as 100 per cent, then it is reasonable to suppose that a fair number occur which do not come to the attention of the attending physician. This becomes more reasonable when upon investigation it is found that these ulcers rarely become chronic but either perforate or heal.

The mechanism of its production is still unknown. Numerous theories have been advanced. Curling[26] believed hyperactivity of Brunner's glands to be responsible. Hunter[28] thought emboli to be the etiologic factor. Many others have attempted the explanation but with inadequate proof. The more recent explanation of the ulcerations being caused in some manner by suprarenal damage deserves mention.[29, 30, 31] Many have attempted the explanation from this viewpoint but so far the proof is not convincing.

Treatment of the Acute Stage.—The treatment of severe burns is very properly divided into the local and the systemic. In reviewing the literature one immediately notices that the controversy seems to center for the most part around the local treatment. Much has been written proving or disproving the value of the tannic acid, the débridement, or the gentian violet treatment of burns, and it is the rule rather than the exception to find the systemic treatment dismissed very briefly. The author wishes to state emphatically that he believes the systemic treatment to be the main pillar in the therapy of burns, and that the variations of the local treatment are, by comparison, emphasized far too much.

The systemic therapy of burns consists in actively combating the primary shock and in anticipating and restoring the fluid loss certain to follow the injury through increased capillary permeability. The treatment of the primary shock is the judicious employment of opiates to keep the patient as free as possible from pain and to promote tranquillity of the mind. Heat in the form of the electric crib is used to combat heat dissipation and to restore normal temperature. The head of the bed is lowered and an intra-

venous injection of isotonic saline is given to combat the relative reduction of the total blood volume. The amount of saline injected intravenously during the stage of primary shock should be a quantity equal in amount to the patient's total blood volume. In a man weighing 150 pounds it is thought this would amount to 5,000 cc. By rapidly injecting this amount of saline intravenously, the relative blood volume is apparently restored. When the tone of the capillary bed recovers, the circulation will have an excess quantity

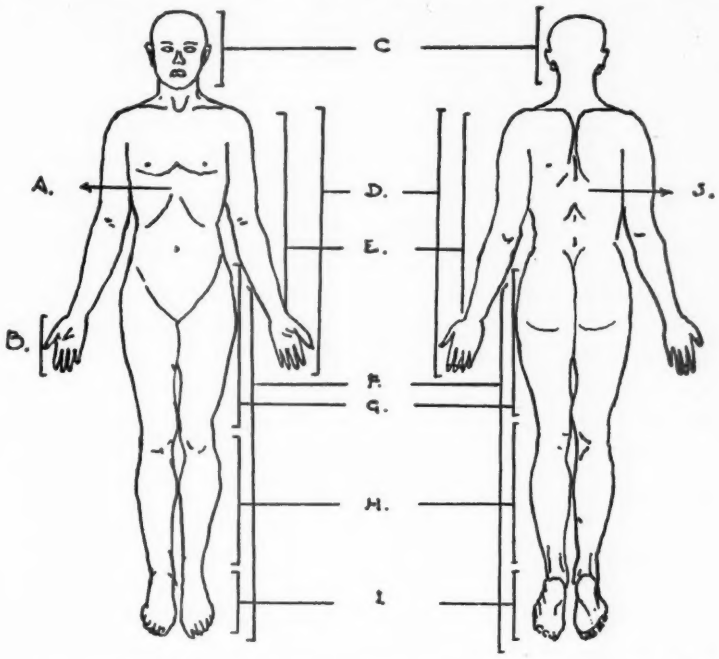


FIG. 1.—Showing method of estimating the percentage of the area burned by use of the Berkow scale.

A—Anterior surface of trunk	20%
B—Total surface both hands	4½%
C—Head	6%
D—Total surface, upper extremities complete	18%
E—Total surface, both arms and forearms	13½%
F—Total surface, lower extremities complete	38%
G—Total surface, both thighs	19%
H—Total surface, both legs	13⅔%
I—Total surface, both feet	6⅓%

To ascertain the proportions at a given age, the following rule is proposed: the trunk is 40 per cent, the upper extremities, 16 per cent. For the head and lower extremities, subtract the age (in years) from 12 and add the remainder to the number expressing the adult proportion for the head (6 per cent). Subtract the same amount from the number expressing the adult proportion (38 per cent) for the lower extremities.

of fluid present to combat the gradual loss of serum now to ensue due to the increased capillary permeability. These measures should successfully combat the primary shock. The restoration of the fluid lost from increased capillary permeability is merely a question of the quantity and kinds of fluid one should give the patient. The quantity is determined in two ways, first, the surface area involved, and, secondly, the variations of the red blood cell count and hemoglobin index. Bearing in mind the experiments of Under-

hill, that an animal with one-sixth of the body surface burned would lose in the first 24 hours fluid equivalent to 70 per cent of its total blood volume and that this in an adult weighing 150 pounds would amount to 3,500 cc., one has a basis for quantitative estimation. As has been stated before, the fluid loss is directly proportionate to the surface area involved. A rough estimate of the area burned can be made by using the Berkow scale[32] (Fig. 1). By following the red blood cell count and the hemoglobin index taken each time from the same site one has a much simpler way of estimating the degree of blood concentration and the patient's fluid requirements. In following the latter method the general rule should be to force fluids, no matter to what quantity, until the blood concentration is restored to normality. The fluids should be subsequently forced in sufficient quantities to maintain blood concentration within normal limits. During the first 48 hours following a burn, when it can be reasonably assumed that the patient's heart and kidneys are functionally normal, there is no danger in forcing isotonic fluids in any quantity. This has been adequately proven by Smith and Mendel[33] who have shown that the excess fluid is eliminated by the kidneys without harm to the animal organism as rapidly as it is possible to inject it. Since an extensive burn renders subcutaneous injection of fluids almost impossible the intravenous route is the method used almost entirely. The oral and rectal routes, however, should not be neglected.

The kinds of fluids to be used present another problem. Since Underhill and his associates have proven that the fluid lost is almost analogous to blood serum, it is obvious that the ideal fluid would be blood serum. To give this fluid in the quantity required would be obviously impossible. The normal saline solution approaches nearer the nature of blood serum than any other fluids commonly used.[34] It contains the water necessary to restore blood volume. It contains the sodium and chlorine ions so important to the maintenance of the acid-base equilibrium and the electronic concentration.[35] However, it must be emphasized again that saline solution alone is inadequate to the proper restoration of blood volume. The serum protein lost in the burned area must be restored in order that the colloidal content of the blood stream will be sufficiently high to prevent the saline injected from diffusing out into the tissue spaces.[23] Too often we see the burned patient die with a concentrated blood while his peritoneal cavity shows free fluid at postmortem. Furthermore, reduction of the serum protein content of the blood has in itself been proven to be dangerous and even fatal.[36] The simplest way to restore serum protein lost is by blood transfusion. At least 1,000 cc. of blood should be given to adult patients in the first 24 hours and, in the extensively burned case, more than this should be given if it is feasible.

In analyzing the various methods used in the local treatment of burns one finds that the controversy is more apparent than real. The clinics advocating the tannic acid treatment admit the possibility of infection and in using tannic acid sprays to combat the absorption of the tissue toxin and also to prevent the escape of plasma and water they consciously or unconsciously

practice the chemical débridement taught by the bacterial toxin school. Thus it is easily possible for one to believe in the bacterial toxin theory and still use the tannic acid spray. On the other hand, the clinics advocating the bacterial toxin theory use gentian violet solution as a bactericide and in doing so precipitate protein which forms an eschar. This eschar is likewise chemical débridement and would also retard the absorption of a tissue toxin should it be present. It is easy to see, therefore, why so many authors avoid controversy by admitting the possibility of both toxins because, in the practical treatment of burns, either tannic acid or gentian violet can be used irrespective of which type of toxin one believes in. If, on the other hand, the clinician believes in the existence of neither toxin, and believes that the secondary shock stage of burns is purely the result of fluid loss, there still remain reasons why he should use either the tannic acid or the gentian violet spray. Firstly, the use of either of these sprays will form an eschar which decreases pain. Secondly, it is easily possible that the precipitation of protein to form the eschar may lessen fluid loss by the early establishment of the electronic balance. Thirdly, the chemical débridement caused by either solution lessens the possibility of bacterial contamination becoming an infection and is far superior to any form of mechanical débridement in preventing the third (infectious) stage of burns.

In the local treatment of burns the author believes that, whereas either tannic acid or gentian violet may be used, the gentian violet solution has certain advantages which should be considered. Both tannic acid and gentian violet solution will precipitate protein to form an eschar which decreases pain, chemically débrides, and lessens absorption, but gentian violet is more bactericidal than tannic acid, and the eschar formed is more soft and pliable. Particular attention should be paid to this latter advantage as it permits the early palpation of accumulations of pus beneath the eschar and easy evacuation. The early evacuation of pus pockets beneath the eschar protects the epithelium of the hair follicles, and the sebaceous and sweat glands from destruction. As it has been adequately proven that regeneration of skin takes place from these sources, this is obviously of great importance in the reconstruction phase.[37]

The local treatment begins with the admission of the patient. The burned area should be cleansed of all foreign matter and mechanical débridement carried out as far as is compatible with the patient's general condition. The burned case should be placed upon sterile sheets beneath an electric crib and either the tannic acid or gentian violet spray used. If the tannic acid spray is to be used the aqueous solution should be freshly prepared. The strength varies from 2.5 per cent as originally used by Davidson[11] to 10 per cent as used by Mason[38] and Mitchiner.[12] The spray is used every 15 minutes until a brownish-black eschar is formed and then every hour until 48 hours have passed. If the gentian violet spray is used the solution should be 1 per cent in water and applied every one to two hours until the eschar is formed, then every four to six hours until healing is complete.[22] Fatherree,

Kennedy and McSwain[39] have altered this treatment by making a gentian violet jelly which is spread upon sterile cloths and applied. The jelly consists of 1,000 cc. of a 1 per cent aqueous solution of gentian violet and 30 Gm. of tragacanth. This jelly, it is claimed, rarely has to be applied more than once. In situations where adequate nursing care is impossible this modification may be considered. Irrespective of which solution is used the eschar should be watched carefully after 48 hours for accumulations of pus and such accumulations promptly evacuated and Dakin's dressings applied.

SUMMARY

(1) The various theories concerning the ill effects of burns have been analyzed and an attempt has been made to show that the phenomena associated with burns are in reality a sequence of events.

(2) The pathology and complications of burns have been discussed.

(3) The treatment of severe burns has been given on the basis that the associated phenomena are a sequence of events. The treatment has, furthermore, taken into consideration the fact that any or all of the various theories concerning burns may be correct.

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THE BLEEDING VOLUME IN SEVERE BURNS

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ROOME, KEITH, and PHEMISTER[1] showed that the methods by which experimental shock could be produced may be divided into two groups, dependent on the bleeding volume. In the first type, produced by hyperventilation, anaphylaxis, histamine administration, spinal cord section, or spinal anesthesia, the bleeding volume was large. In 28 dogs it averaged 49. 9 per cent of the calculated blood volume. This is only slightly less than the average of 58. 6 per cent obtained for a series of 20 control dogs. On the other hand, in the second group, due to trauma to an extremity, hemorrhage, plasmapheresis, or intestinal manipulation, the bleeding volume was greatly reduced, averaging 21.8 per per cent in 22 dogs. The blood pressure was reduced in the 50 shock experiments to levels usually between 50 and 70 Mm. of mercury and the animals were usually bled to death approximately one hour after the initiation of the procedure. The 20 anesthetized control dogs were bled to death at the end of one hour. The action is in the first group mainly the result of peripheral vasodilatation and in the second group mainly of reduction in circulating blood volume.

This differentiation of shock into two types on the basis of bleeding volume and blood pressure is in accord with the work of Blalock and his associates on the basis of cardiac output and blood pressure. Blalock[2] attempted to produce primary shock by striking the abdomens of ten well-anesthetized dogs many blows in rapid succession with the flat surface of a board for approximately two minutes. If free hemorrhage into the peritoneal cavity resulted from the traumatization, the experiment was discarded. In all ten animals the striking of the abdomen was followed by a decline of blood pressure varying from 38 to 75 Mm. of mercury. Associated with the drop in blood pressure, the cardiac output as measured by the Fick method increased slightly in seven animals and decreased in three. Johnson and Blalock[3] found that the introduction of histamine causes an initial decline in the blood pressure followed by a decrease in the cardiac output. Blalock and Beard[4] found that the removal of the adrenal glands usually causes a decline in blood pressure before the cardiac output is lowered. Blalock and Bradburn[5] found that trauma to the central nervous system is usually associated with a simultaneous decline in both the cardiac output and blood pressure. In addition, Butler, Beard, and Blalock[6] found that following the subcutaneous injection of histamine in dogs there was a decrease in the blood volume as determined by the dye method. Blalock's

results in general indicate that in primary types of shock, the cardiac output does not decrease until the blood pressure falls. The time relation between these two factors and the blood volume has not yet been worked out completely.

In the so called secondary types of shock, Blalock and his co-workers have found that in general the decrease in cardiac output precedes the decline in blood pressure. Blalock[7] found that repeated removal of blood was usually associated with a decline in the cardiac output from 30 to 50 per cent below the normal level before a marked diminution in the blood pressure occurs. Johnson and Blalock[3] found that secondary shock as a result of trauma to muscles, trauma to the intestines, or burns is associated with a definite decrease in the cardiac output before the blood pressure falls appreciably. No reports of the bleeding volume in experimental burns have been found.

METHOD

Dogs under complete anesthesia were used in all experiments and none that were burned were allowed to recover. They were weighed after catheterizing the urinary bladder and anesthetized with sodium barbital (260 mg. per kg. body weight intraperitoneally). The hair was removed from the body and extremities of the animals by the use of either fine clippers or a razor. The longitudinal midlines of both the dorsal and ventral surfaces were marked with India ink. The blood pressure was determined by placing in the carotid artery a cannula which was connected to a mercury manometer. Hemoglobin determinations were made by the Sahli method and hematocrit readings with the Van Allen hematocrit. The animals were then burned over a period varying from six to ten minutes by the direct application of the Bunsen flame. Only one side, usually the right, was burned with most of the burning over the region of the thigh, groin, axilla and shoulder. The blood pressure was determined intermittently until it had fallen to the desired level. The animals were then bled rapidly to death and the amount of blood obtained was measured. Bleeding was by the carotid arterial cannula, the animal being left in the horizontal position and the cannula kept free from clots. In all animals, tipping of the hind end up after bleeding had ceased caused no more blood to flow. In certain animals cannulization of the other carotid artery after bleeding had ceased caused no more blood to flow. The bleeding volume thus obtained is presumed to be an approximate index of the circulating blood volume, and will be described in this paper as a percentage of the calculated blood volume (1/13th of the body weight). The entire animal was then weighed to determine the total weight loss. This total weight loss was then corrected so as not to include the blood lost by bleeding and any urine or feces which were passed during the experiment. The animal was then bisected up to the neck by the method described in the first paper of this series,[8] the two halves weighed and the

TABLE I

Summary of Results in Six Burned Animals in Which the Blood Pressure was Allowed to Fall Near a So Called Shock Level before the Bleeding Volume Was Obtained. Animal No. 3 Was Burned an Additional Five Minutes at the End of 13½ hours

Experiment	Time of Observation after Burning	Weight of Animal kg.	Loss in Weight in Gm./kg./hr.	Mean Blood Pressure Mm. of Hg.	Hemoglobin Per Cent	Hematocrit	Weight Burned Side Animal kg.	Weight Unburned Side Animal kg.	Difference in Weight Two Sides as Per Cent Body Weight	Terminal Bleeding Volume, Per Cent of Calculated Blood Volume
1	Control period	10.408	—	100	105	53	—	—	—	—
	5 hours	—	—	102	—	—	—	—	—	—
	10 hours	—	—	100	118	60	—	—	—	—
	17½ hours	10.118	1.5	50	119	64	3.246	2.933	3.0	11.9
2	Control period	9.212	—	148	111	53	—	—	—	—
	4 hours	—	—	110	—	—	—	—	—	—
	9 hours	—	—	96	132	62	—	—	—	—
	16 hours	8.746	3.2	44	134	64	2.813	2.583	2.5	14.6
3	Control period	7.835	—	116	110	53	—	—	—	—
	3 hours	—	—	84	—	—	—	—	—	—
	4½ hours	—	—	88	—	—	—	—	—	—
	7 hours	—	—	84	108	53	—	—	—	—
	8½ hours	—	—	86	—	—	—	—	—	—
	13½ hours	—	—	96	—	—	—	—	—	—
	16 hours	—	—	100	—	—	—	—	—	—
	17 hours	7.365	3.5	48	126	65	1.950	1.927	0.3	16.1

4	Control period	10.493	—	164	99	47	—	—	—	—
	4½ hours	—	—	162	130	58	—	—	—	—
	11½ hours	—	—	142	130	64	—	—	—	—
	15 hours	—	—	116	139	62	—	—	—	—
	19½ hours	—	—	82	130	63	—	—	—	—
	21½ hours	10.267	1.0	62	125	60	3.327	3.216	1.1	16.7
5	Control period	12.086	—	148	104	49	—	—	—	—
	3½ hours	—	—	140	140	64	—	—	—	—
	11 hours	—	—	126	153	69	—	—	—	—
	14 hours	—	—	114	154	70	—	—	—	—
	19 hours	—	—	90	156	72	—	—	—	—
	21 hours	—	—	86	—	—	—	—	—	—
	22½ hours	—	—	84	—	—	—	—	—	—
	24 hours	11.482	2.2	82	162	70	3.898	3.518	3.0	14.0
6	Control period	6.855	—	118	106	48	—	—	—	—
	6 hours	—	—	120	132	66	—	—	—	—
	10½ hours	—	—	110	131	66	—	—	—	—
	14 hours	—	—	88	137	68	—	—	—	—
	15½ hours	—	—	80	—	—	—	—	—	—
	16 hours	—	—	76	—	—	—	—	—	—
	18 hours	6.712	1.2	62	134	68	2.211	2.047	2.4	24.6
Average		—	2.1	—	—	—	—	—	2.1	16.3

difference in weight obtained. Control animals were treated exactly the same except that they were not burned.

RESULTS

Fifteen experiments were performed. These represent three groups: (1) Six burned animals in which the blood pressure was allowed to fall to near a so called shock level (varying from 44 to 82 Mm. of mercury) before the bleeding volume was determined; (2) five burned animals in which the bleeding volume was determined before the blood pressure had fallen near a so called shock level; and (3) four control animals. The results of these three groups of experiments are shown in Tables I, II and III. These results are discussed under the following six heads: (*a*) Duration of experiment; (*b*) total weight loss during experiment; (*c*) fall in mean arterial blood pressure; (*d*) rise in hemoglobin percentage and hematocrit reading; (*e*) local accumulation of fluid in the burned tissues as shown by the difference in weight of the burned and unburned sides; and (*f*) the terminal bleeding volume in terms of per cent of the calculated blood volume.

(*a*) *Duration of Experiment.*—The time necessary to reduce the blood pressure to approximately a shock level varied in six dogs from 16 to 24 hours, averaging 17.3 hours. This places the reaction well within the confines of secondary shock, irrespective of whether the time or the etiologic definition of secondary shock is used. The time that was allowed to elapse in the case of the five animals that were bled before the blood pressure had fallen to near a shock level varied from five to 24 hours, averaging 11 hours. Since, in the experiments of Roome, Keith and Phemister,[1] the bleeding volume in both the shock and control animals was usually determined only an hour after the initiation of the procedure, it was thought necessary to determine the bleeding volume of control dogs after a longer interval. Hence, four anesthetized control animals were bled from 15½ to 24 hours, averaging 18.9 hours after the beginning of the experiment. The bleeding volume of these four control animals averaged only slightly less than that of Roome, Keith and Phemister's ten dogs bled at the end of only an hour. Also because the present experiments were of longer duration than those of the authors mentioned, it is not strictly accurate to compare the bleeding volume in burn shock with that in other types of shock as determined in their paper.

(*b*) *Total Weight Loss During Experiment.*—Roome and Wilson[9] showed that the weight loss in five control dogs, observed over a period of 46.2 hours, amounted to 1.9 Gm. per kg. body weight per hour. It was shown in a previous paper[8] that the weight loss in three burned animals observed over a period of from four to ten hours averaged 2.5 Gms. per kg. per hour. In Table I it is seen that in six burned animals observed over a period of from 16 to 24 hours the total weight loss averaged 2.1 Gm. per kg. per hour. The single reading shown in Table II raises this average to 2.2 Gm. per kg. per hour for seven burned animals. This agrees rather well

TABLE II

Summary of Results in Five Burned Animals in Which the Bleeding Volume Was Determined before the Blood Pressure Had Fallen Near a So Called Shock Level. Animal No. 7 Was Burned an Additional Five Minutes after 14½ Hours, Three Minutes after 22 Hours and 13 Minutes after 24 Hours. The Fluid Shift Shown in the Next to the Last Column for Experiments No. 8 and No. 9 Is Not Included in the Average Because Part of the Fluid Had Leaked Out of the Burned Tissues before Weighing

Experiment	Time of Observation after Burning	Weight of Animal kg.	Loss in Weight in Gm./kg./hr.	Mean Blood Pressure Mm. of Hg.	Hemoglobin Per Cent	Hematocrit	Weight Burned Side Animal kg.	Weight Unburned Side Animal kg.	Difference in Weight Two Sides as Per Cent Body Weight	Terminal Bleeding Volume, Per Cent of Calculated Blood Volume
7	Control period	11.255	—	145	106	49	—	—	—	—
	3½ hours	—	—	150	—	—	—	—	—	—
	8 hours	—	—	140	149	67	—	—	—	—
	14½ hours	—	—	116	141	64	—	—	—	—
	20 hours	—	—	106	—	—	—	—	—	—
	22 hours	—	—	92	—	—	—	—	—	—
	23 hours	—	—	102	—	—	—	—	—	—
	24 hours	—	—	108	149	70	3.475	3.225	2.2	29.3
8	Control period	8.455	—	140	106	49	—	—	—	—
	5½ hours	8.331	2.7	106	126	63	2.422	2.319	1.2	49.2
9	Control period	14.0	—	140	99	46	—	—	—	—
	3 hours	—	—	136	148	68	—	—	—	—
	5 hours	—	—	114	132	63	4.217	4.052	1.2	22.3
10	Control period	24.5	—	132	105	51	—	—	—	—
	2½ hours	—	—	126	126	58	—	—	—	—
	6½ hours	—	—	114	131	66	—	—	—	—
	10½ hours	—	—	102	132	58	8.585	7.788	3.3	21.1
11	Control period	25.0	—	140	98	45	—	—	—	—
	1½ hours	—	—	130	103	48	—	—	—	—
	6 hours	—	—	138	112	54	—	—	—	—
	10 hours	—	—	130	146	—	8.516	7.649	3.5	35.4
Average		—	2.7	—	—	—	—	—	3.0	31.4

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TABLE III

Summary of Results in Four Anesthetized Control Animals that Were Not Burned

Experiment	Time of Observation	Weight of Animal kg.	Loss in Weight in Gm./kg./hr.	Mean Blood Pressure Mm. of Hg.	Hemoglobin Per Cent	Hematocrit	Weight Right Side Animal kg.	Weight Left Side Animal kg.	Difference in Weight Two Sides as Per Cent Body Weight	Terminal Bleeding Volume, Per Cent of Calculated Blood Volume
12	Onset	24.0	—	154	86	40	—	—	—	—
	10 hours	—	—	142	88	42	—	—	—	—
	14½ hours	—	—	128	78	40	—	—	—	—
	24 hours	—	—	128	67	33	6.948	6.920	0.12	49.8
13	Onset	12.975	—	134	109	54	—	—	—	—
	9½ hours	—	—	138	113	52	—	—	—	—
	14 hours	—	—	132	103	49	—	—	—	—
	17½ hours	12.711	1.2	136	103	49	—	—	—	52.0
14	Onset	12.070	—	136	112	48	—	—	—	—
	9 hours	—	—	130	113	49	—	—	—	—
	13½ hours	—	—	120	112	47	—	—	—	—
	15½ hours	11.922	0.8	102	91	41	3.643	3.620	0.19	47.2
15	Onset	19.5	—	118	85	43	—	—	—	—
	9 hours	—	—	124	84	41	—	—	—	—
	18½ hours	—	—	116	91	42	—	—	—	64.7
Average		—	1.0	—	—	—	—	—	0.16	53.4

with the figure of 2.5 reported in the previous paper. The average of 1.0 Gm. per kg. per hour for two control animals shown in Table III agrees fairly well with the figure of Roome and Wilson. The importance of these figures is that they indicate there is little more weight loss from all causes in the burned than in the unburned control animals. This would in turn support the theory that in dogs an internal fluid imbalance is the important factor in the causation of burn shock. In the human being there is the additional factor of weeping from the burned skin, whereas a dog's skin is essentially tanned and dry immediately after burning.

(*c*) *Fall in Mean Arterial Blood Pressure.*—In the six experiments shown in Table I, the mean arterial blood pressure was allowed to fall to 50, 44, 48, 62, 82, and 62 Mm. of mercury respectively. All but the reading of 82 is below the so called shock level of blood pressure (70 Mm. of mercury). The average blood pressure at the end of the six experiments was 58. Since the average initial blood pressure in these six experiments was 133, the final reading represents an average fall of 75 Mm. of mercury. In the five experiments in which the bleeding volume was determined before the blood pressure had fallen near a so called shock level, the average initial mean arterial blood pressure was 141 and the average final reading was 113, representing a fall of 28 Mm. of mercury. In the four anesthetized control dogs that were not burned the average initial pressure was 135, the average final pressure 120, and the average fall 15 Mm. of mercury.

(*d*) *Rise in Hemoglobin Percentage and Hematocrit Reading.*—In the six burned animals that were bled after the blood pressure had fallen appreciably, the average readings at onset were 106 per cent for the hemoglobin and 50 per cent for the hematocrit. The highest readings after burning averaged 136 and 66 which represent an increase of 30 in the per cent of hemoglobin and 16 in the hematocrit readings. Similar figures from Table II are 103 and 48 at the onset, and 140 and 67 for the highest readings, representing a rise of 37 in the hemoglobin per cent and 19 in the hematocrit reading. The average hemoglobin per cent in the control experiments shown in Table III did not rise appreciably at any time, the initial reading averaging 98 and the final 88, representing a fall of 10. The hematocrit readings in these control animals showed a similar average drop of five from 46 to 41. In the nine burned animals in which sufficient readings were made to follow the course of the hemoglobin percentage, three showed a terminal decrease, five showed a terminal rise and one showed no appreciable change at the end. The hematocrit readings in general followed the hemoglobin percentage. It is possible that if readings were made more frequently a higher proportion of experiments would have shown the terminal drop.

(*e*) *Local Accumulation of Fluid in the Burned Tissues as Shown by Difference in Weight of the Burned and Unburned Sides.*—The amount of this accumulation was determined so as to have an index of the extent of the burn. Blalock[10] showed that in nine burned dogs the fluid amounted to 3.2 per cent body weight at death. In a previous paper in this series, the

fluid in eight burned animals averaged 2.2 per cent body weight at death. In Table I it is seen that the difference in weight of the sides of the animals averaged 2.1 per cent body weight. The very low reading of 0.3 per cent in experiment three is probably due to the inaccuracy of the bisection method combined with a relatively slight burn as is evidenced by the slight changes in the hemoglobin and hematocrit readings. In three experiments shown in Table II it averaged 3.0 per cent. On the other hand, two control experiments shown in Table III showed a practically negligible difference of 0.16 per cent body weight. The figures for the burned animals indicate a fluid shift of only slightly less magnitude than that obtained by Blalock.

(*f*) *The Terminal Bleeding Volume in Terms of Per Cent of the Calculated Blood Volume.*—The average terminal bleeding volume in the six burned animals in which the blood pressure was allowed to fall near a so called shock level before bleeding averaged 16.3 per cent of the calculated blood volume (one-thirteenth of the body weight). The similar average for the five intermediate experiments shown in Table II was 31.4 per cent. The average bleeding volume for the four control dogs was 53.4 per cent. The decrease in bleeding volume in the first series of six animals shown in Table I was 37.1 per cent and at the same time the blood pressure fell an average of 75 Mm. of mercury. The decrease in bleeding volume in the second or intermediate series of five animals shown in Table II was 22.0 and at the same time the blood pressure fell an average of 28 Mm. of mercury. This indicates that at a time (as shown by the second series of five animals) when the blood pressure had fallen only 37 per cent of the ultimate fall (as shown by the first series of animals), the bleeding volume had already decreased 59 per cent of the ultimate amount. It is, of course, difficult to compare the results of these two series of animals. It can only be assumed that if the blood pressures of the second series of five animals had been allowed to fall as low as in the first series of six animals, the bleeding volumes would have decreased the same.

COMMENT

Five of the most important physiologic changes in burns are those in (1) blood pressure, (2) blood concentration as shown by increase in per cent hemoglobin and hematocrit reading, (3) local fluid accumulation in the tissues, (4) blood volume, and (5) cardiac output. The time relations of these five factors are of interest. The work of previous investigators[3, 8, 10, 11] has shown that in general there is a gradual increase in local fluid accumulation, decrease in cardiac output and increase in blood concentration following experimental burns. On the other hand, the blood pressure remains near normal only to collapse rather suddenly as death approaches. Little work has been done on the blood volume following burns. This paper involves the assumption that the bleeding volume is an index of the circulating blood volume. Whether or not this assumption is justified is an open question, but the bleeding volume itself is an index of the type of shock in a given instance

as was shown by Roome, Keith, and Phemister.[1] These authors studied the bleeding volume in most of the important types of experimental shock except burns. Blalock has studied the cardiac output[3] in burns and the blood volume[6] in certain types of shock other than burns. The average bleeding volume in the 11 burned dogs was 23.2 per cent of the calculated blood volume. This agrees rather well with the figure of 21.8 obtained by Roome, Keith, and Phemister[1] for the bleeding volume in shock due to trauma to an extremity, hemorrhage, plasmapheresis, or intestinal manipulation in all of which blood volume is reduced. This would indicate that the shock resulting from burns is similar to that following these other procedures (using the bleeding volume as the basis of comparison). All of these injuries are generally recognized as producing secondary shock. On the other hand, Roome, Keith, and Phemister's average of 49.9 per cent of the calculated blood volume obtained for the bleeding volume after hyperventilation, anaphylaxis, histamine administration, high spinal cord section or spinal anesthesia indicates that these former procedures produce a type of shock dissimilar to that resulting from burns.

A comparison of the two series of burned animals supports the belief that the bleeding volume decreases more rapidly than the blood pressure following a severe burn. This agrees with Blalock's finding that bleeding gives reduced blood volume and diminished cardiac output before the fall in blood pressure. The five animals in which the blood pressure had fallen only 37 per cent of that of the other series of six animals showed a fall in bleeding volume which was 59 per cent of that of the other series. This suggests that if Roome, Keith, and Phemister[1] had determined some of their bleeding volumes before the blood pressure had fallen appreciably, they might also have found them low in shock produced by the various other causes in this group.

These changes in the bleeding volume in burns indicate that the blood concentration, local fluid accumulation, blood volume (as evidenced by the bleeding volume) and the cardiac output all follow the true course of a burn more accurately than the blood pressure which collapses only as death approaches. This suggests that clinically any of these factors may be of more importance in diagnosis and prognosis than the blood pressure. The local fluid accumulation, blood volume (most certainly the bleeding volume), and cardiac output are all difficult to determine in the human being; but the hemoglobin percentage and hematocrit reading are easy to obtain and should be more often used in the diagnosis and prognosis of burn shock. The hemoglobin percentage has been especially used in this connection by Underhill.[12]

SUMMARY

The bleeding volume in six burned dogs bled after the mean arterial blood pressure had fallen from an average of 133 to 58 (a drop of 75 Mm. of mercury) was 16.3 per cent of the calculated blood volume. At the same time the animals showed a fluid shift to the burned side of 2.1 per cent of

the body weight. The hemoglobin percentage rose from an average of 106 to 136 and the hematocrit reading from 50 to 66.

The bleeding volume in a second series of five burned animals after the mean arterial blood pressure had fallen from an average of 141 to only 113 (a drop of 28 Mm. of mercury) was 31.4 per cent of the calculated blood volume. At the same time these animals showed a fluid shift to the burned side of 3.0 per cent of the body weight. The hemoglobin percentage rose from an average of 103 to 140 and the hematocrit reading from 48 to 67.

The bleeding volume in four anesthetized control animals averaged 53.4 per cent of the calculated blood volume. The changes in the blood pressure, fluid shift, hemoglobin percentage, and hematocrit reading were all much less than those in the burned animals.

These results indicate that the bleeding volume decreases more rapidly than the blood pressure and that its rate of change is more closely related to the cardiac output, blood concentration, and local fluid accumulation. The average bleeding volume following burns is similar to that obtained by other authors in shock due to trauma to an extremity, hemorrhage, plasmapheresis, or intestinal manipulation in which the chief factor is a decrease in blood volume.

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PILONIDAL CYSTS AND SINUSES

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IT IS our purpose in this paper to present and analyze a series of 288 cases of pilonidal cysts and sinuses with especial reference to treatment and end-results. Pilonidal, sacroccygeal or dermoid cyst, postanal dimple, or foveola coccygea, are congenital malformations in the sacrococcygeal region characterized by acute and chronic inflammation; the presence of tufts of hair; occasional islets of squamous epithelium and foreign body giant cells.

The first description in the literature was given by Anderson[1] in 1847, who reported a case of "hair extracted from an ulcer." Vaughn, quoted by Markoe and Schley,[2] reported three cases of "pilous cysts" in 1865. Warren[3, 4] in 1867 named the condition pilonidal cyst, although he had described some cases previously in 1854. They were mentioned and described as medical curiosities, frequently, a few cases at a time, from this time on, but practically all the literature up to 1900 dealt with the embryologic and etiologic aspects of the condition, with little emphasis on treatment, although Gussenbauer[5] recommended excision by thermocautery as early as 1893.

All commentators with the exception of one early writer (who considered them due to ingrowing hairs) agree that the conditions are of congenital origin. The theories, however, vary widely as to the particular embryonal layer from which they arise, as well as their mode of origin. Walker[25] believed his case to be a "monstrosity by inclusion." Feré[26] and Bland Sutton[6] regarded sinuses and cysts as "slight defects in the coalescence of the superficial portions of the medullary folds in the sacrococcygeal region." Terrillon[7] stated they were a special form of spina bifida involving the skin. Lawson Tait[8] suggested that the coccygeal dimple is the "cicatrix of the spina bifida by which the human tail has been lost." Lannelongue[9, 10] suggested the following interesting theory: "The mesoblast lies, after the formation of the medullary canal, between the external epidermis and the vertebral column, except in the region of the sacrum, where little of this tissue is interposed, so that this region is reduced to epidermis and bone. Consequently, the superficial layer, the epiblast, joined at a later period to the mesoblast, preserves closer relation with the bone, and later, when the subcutaneous tissue is developed around these places a depression will be formed. If deep and narrow enough, the orifice may close up and a dermoid cyst will be the consequence." Tourneaux and Hermann,[11] who worked with the greatest amount of material, concluded that the sinuses and cysts were remnants of the conflict for survival

of the medullary tube as encroached on by the last portions of the spine to close, believing they were epidermis invaginated by the pull of the filum terminale. The foveola coccygea (or fossette medullaires) *i.e.*, the English "dimple," they attributed to the inturning of the coccyx. They could find no evidence of contact with the postanal gut.

Mallory[12] in America, after dissection of six fetuses, confirmed the work of Tourneaux and Hermann[11] as to remnants of the medullary canal, and believed these capable of giving rise to the picture seen in congenital sinuses and cysts. He stated that in all probability it is only these congenital sinuses and cysts that give rise to suppurative sinuses. Most French and German authors lean to the views developed by Lannelongue.[9] Aschoff[13] also threw the weight of his authority to the view that sinuses and cysts are due to faulty median skin agglutination of the sacrococcygeal region, although he agreed with Tourneaux and Hermann[11] that the foveola coccygea are due to anteroflexion of the coccyx in which the remaining strands of the medullary tube act as tractors on the skin. Nassè[14] mentions the neurenteric canal as a possible origin, and thinks all cysts can be explained as arising from this or the medullary canal. Klemm,[15] while attributing the cysts to faulty skin development, believed that after invagination, growth of the invaginated skin occurs to produce the multiple sinuses.

Masson[16] quotes Ewing as stating that the embryonal structure which gives rise to all growths in this region are chiefly: (1) Fovea coccygeal and coccygeal vestiges of the neural canal. (2) Neurenteric canal. (3) Postanal gut. (4) Proctodeal membrane. To these must be added the epithelium of the sacrococcygeal region. All commentators agree, however, that the cysts, sinuses and dimples of the sacrococcygeal region arise from one primitive layer only.

Against the theory of inclusion bodies are the facts: (1) That hair and squamous epithelium only of the possible teratoid structures are found constantly in these conditions. (2) The structures present are fully differentiated. (3) Malignant change has never been described as occurring in a definite pilonidal cyst.

Between the structures under discussion and the various tumors of this region there are in the case of the first only true skin formations, whereas in the latter all types of epithelial structures are found as well as connective and nervous tissue. If we are to assume that the neurenteric canal or neural groove or medullary canal have anything to do with the formation of cysts or sinuses, we must explain why only true skin tissues are ever found in these latter structures. One would certainly expect to find some transition from columnar to squamous epithelium. One has also to explain why nerve tissue is notably absent in cysts. The rôle of the vestigial structures in the formation of tumors of the sacrococcygeal region is undeniable. They probably never play a rôle in the formation of cysts or sinuses. The dimple may possibly be due to the traction caused by the inturning of the coccyx.

We are led, therefore, to feel that these cysts and sinuses are purely and solely derived from faulty agglutination of fetal skin folds. The presence of similar lesions in other parts of the body in the midline are confirmatory inferentially. Clinically this is confirmed by the findings at operation. It has been found that the sinuses and cysts are in the skin and subcutaneous tissue and invariably are bounded by the deep fascia covering the dorsum of the sacrum and coccyx. In our series it has never been found necessary to incise this covering in order to completely excise these lesions.

In those cases where multiple sinuses are found they are probably due to infection resulting in multiple pointing and rupturing and are as a general rule lined by granulation tissue. Certain cases, however, are said to show multiple epithelium-lined sinuses and if these exist, we must fall back to the theory of Klemm[15] for the explanation. If our explanation is to be accepted

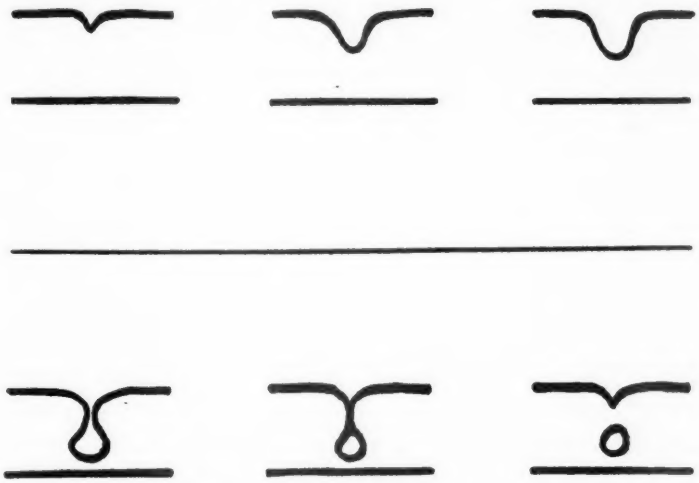


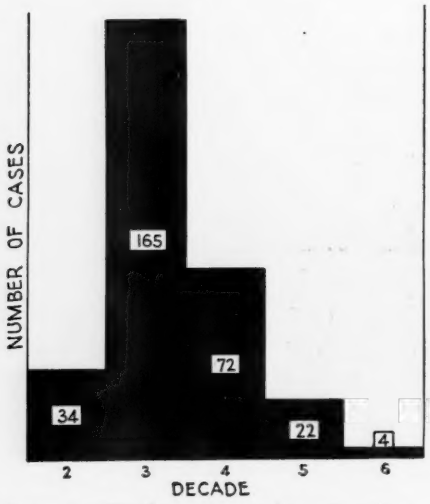
FIG. 1.—Illustrating the formation of the variations found (from Markoe and Schley).

the following simple diagrams taken from Markoe and Schley[2] adequately illustrate the formation of the variations found (Fig. 1).

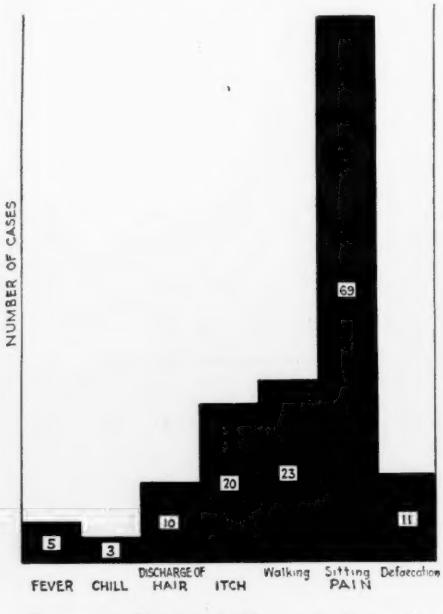
The true microscopic pathology of these conditions is probably rarely seen. The sections of these cases showed no case of simple uninflamed tissue. The predominant picture was acute and chronic inflammation. The peculiarities were numerous foreign body giant cells, and hair for which we could find no hair follicles. No sweat or sebaceous glands were found in the interiors of the cysts and sinuses, which could definitely be said to be connected with them or part of these congenital conditions. So far as our examinations of this large collection of material are concerned, the origin of the hair found in these conditions remains unexplained. Small islands of squamous cells were occasionally found either free or doubtfully attached to the cyst linings. Squamous epithelium containing the normal structures was found dipping into the sinuses at their entrances, but this epithelium became continuous at once with the inflammatory cyst or sinus lining. Harvey Stone[17] had one

clear cut specimen in 61 cases. He notes only: "In a short sinus is a slightly modified invagination of true skin. None of its elements is fully developed."

In the earlier literature Gussenbauer,[5] Crone,[18] describe the presence of squamous epithelium, hair follicles, sebaceous and sweat glands. As neither of them had many cases (Crone[18] had only six), it is curious that they should have seen these things. The origin of the conception that these things are present in sacrococcygeal cysts probably can be traced to these writers. They are not described by pathologists or commentators on much larger series. The most careful microscopic studies made, those of Oehlecker,[19] including serial sections, showed no characteristic epithelial structures.



GRAPH 1.—Showing age groupings of patients on admission.



GRAPH 2.—Showing incidence of the various symptoms on admission.

Incidence.—Winkler[20] found 30 cases in 19,000 hospital admissions or 1 : 600. Wette[21] found seven cases in 15,000 hospital admissions or 1 : 2,143. Our 288 cases were found in 481,384 hospital admissions or 1 : 1,683.

Race.—Thomason[22] in Texas (1934), and Stone[17] in Maryland (1924) found no colored patients in their series. Our series shows one female and three male colored patients.

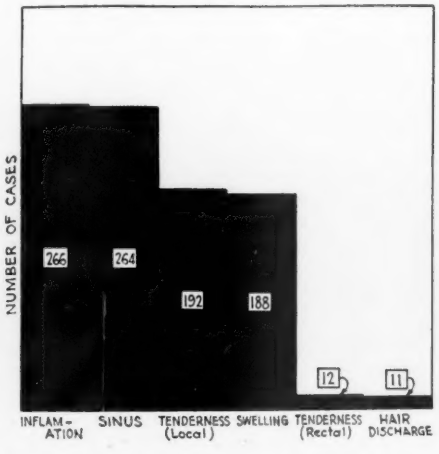
Age.—Depres[23] and Lannelongue[9] state that congenital sacrococcygeal cysts occur in one-third of all new born in some degree, but only 3 per cent of adults show it. Tait[8] stated that they occurred in 23 per cent of all women examined. Over one-half of the cases come for treatment in the third decade, and one-fourth in the fourth decade (Graph 1).

Sex.—In this series 241 were males and 47 females. One might expect this to have some bearing on the question of trauma. Of the 39 cases who

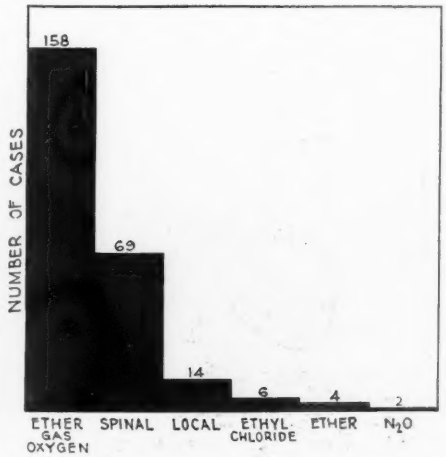
gave histories of trauma, 27 were males, 12 females, the time varying from one week to eight years, the greater number being within one month. They consisted of blows, falls, jolting of cars, *etc.*

Hereditary Factors.—There was no evidence of any familial disposition to these abnormalities, only five cases having a family history of pilonidal cysts and sinuses. Congenital malformation other than sacrococcygeal sinuses and cysts were present in only one case—club feet. Syphilis and tuberculosis: We found no cases of tuberculosis and curiously enough of 123 Wassermanns taken, only seven were positive, a slightly lower incidence than usual for this age grouping.

Symptomatology.—Itching, pain on sitting or walking and the discharge of pus were the predominant symptoms. One child of four had a tumor



GRAPH 3.—Showing incidence of the various physical signs on admission.



GRAPH 4.—Showing types of anesthesia used.

above the anus since birth. The average time of symptoms before admission was 795 days. The shortest period was three days, the longest 8,760 days. Seventy-six cases had symptoms for a period of three years or over (Graph 2).

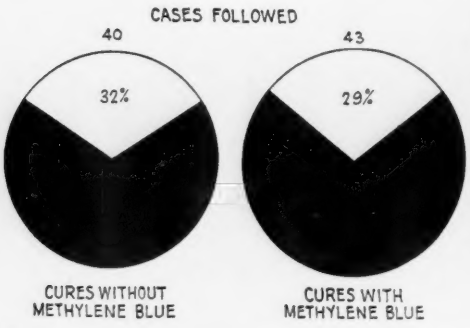
Physical Examination.—All cases in this series showed evidence of inflammation upon admission. It would seem that the simple presence of these abnormalities caused no symptoms important enough to bring the patient to the doctor (Graph 3).

Differential Diagnosis.—Occasionally the following conditions may be considered: Simple acute suppurative inflammation (furuncle), sebaceous cysts, lipomata, fibromata, tuberculosis, chancroid, fungous infections, malignant ulcer, inflammation of congenital tumor, syphilis and anthrax.

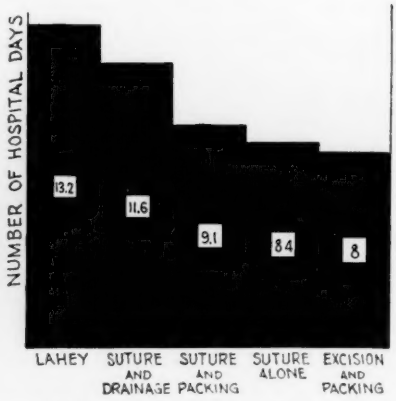
Anesthesia.—In this group, it is noteworthy that only 14 local, and no caudal anesthesias were employed probably because of the juxtaposition of the inflammatory area. Sixty-nine spinal anesthetics were used, probably partially, due to the fact that the renewal of favor to this type of anesthetic

reached its height in the period covered by our series. We feel definitely that the possibility of introducing organisms by spinal puncture, in the presence of an inflammatory lesion of the back, is very great. Gas, oxygen and ether was used in the greater number of cases and would seem to be the method of choice. (Graph 4).

Treatment.—There is unanimity of opinion as to the necessity for complete surgical excision of sacrococcygeal cysts and sinuses for the cure of these conditions. One hundred and six of our cases had one or more previous incisions and drainages. Twenty-nine cases had had previous excision. The method of insuring complete excision and the after treatment of the wounds has been a matter of great debate. The use of a delineating reagent such as methylene blue for outlining the ramifications of the sinuses would seem to be a helpful measure in the excisions. Actually our percentages of



GRAPH 5.—Showing comparative results with or without methylene blue as delineating reagent.



GRAPH 6.—Showing average stay in the hospital postoperatively for all methods of extirpation.

recurrence are the same with or without such a reagent (Graph 5). We have not used roentgen ray of opaque media injections in our series.

Follow Up.—We have been able to follow 83 cases of our series of 288, a rather remarkable percentage of follow up for such a condition, in a general hospital (such as Bellevue) with a large floating population. The follow ups vary from two to 14 months. The methods of extirpation and closure used with their end-results, including the number of postoperative hospital days are shown in Table I. The total cures for the series were 73 per cent for all excisions. Excision and packing show 30 cures for 32 cases. Excision and suture (including plastic procedures) without drainage show 56 per cent cures; excision, suture and packing show 63 per cent cures; excision, suture and drainage show 66 per cent cures, an average of 62 per cent cures for 50 cases in which sutures were used. There was one cure by incision and drainage alone in this series. The postoperative hospitalization for all methods is shown in Graph 6. There were no deaths. There was one case of severe bleeding postoperatively, which, however, did not need transfusion, and one case developed erysipelas. There were five late

complications. One had marked tenderness still at the end of eight months. Two complained of pain about the scar. One note was made of adhesion to the sacrum, and one case formed crusts and failed to epithelialize satisfactorily.

Discussion.—Since all these cases are infected on admission to the hospital and the wounds are in close proximity to the rectum, we have seen practically 100 per cent infection in our follow up observations present during the convalescence in every method used. Thus any method involving closure of the wounds would on general surgical principles seem to be contra-indicated. The tendency to widespread infection is undoubtedly lessened by preliminary incision and drainage of abscesses prior to excision if one be present. The

TABLE I.

Showing End Results for the Various Types of Operation

TYPE OF OPERATION	TOTAL NUMBER OF CASES	NUMBER OF CASES FOLLOWED	NUMBER OF CURES	PERCENT OF CURES
Excision & Packing	132	32	30	94
Excision, Suture & Packing	79	24	15	63
Excision & Suture	29	18	10	56
Excision, Suture & Drainage	27	9	6	66
Incision & Drainage	21	16	1	7
All Types (totals)	288	99	62	63
Corrected For I. & D.	267	83	61	73

probability is that infection will be a less prominent factor if the excisions are really radical. The tendency to try to conserve tissue cannot be considered in the removal of pilonidal cysts and sinuses. The necessity for dressings from six weeks to three months should be understood by the patient from the beginning and this period in our experience has not been shortened by any method of suture attempted, including that of Lahey, in which drainage from the lower angle of the wound has lasted regularly six to eight weeks. The Lahey[24] operation differs from other methods of suturing in that it displaces the scar lateral to the midline, in order to prevent tenderness and pain on sitting. Only three of our cases with midline scars complained of pain over the scar, obviously not enough to warrant the opening of a new area to infection.

We have emphasized the almost universal presence of infection. This is minimized to a great extent by meticulous care and frequent dressings. In the majority of our cases iodoform gauze packing was used as a deodorant,

but antiseptics in general, dichloramine T, potassium permanganate, mercurochrome, *etc.*, have been used only for the first few days. We have not noted any superiority of antiseptics to packing alone.

The dressing is important. It should prevent the seepage of exudate over the anal region and perineum. It should remain in place during defecation, sleep and walking. The following has been found most practical. The patient is dressed in the erect position. The wound is washed with tincture of green soap and peroxide. The wound is packed lightly with a large gauze fluff to completely fill the cavity. A marine or rubber bath sponge about an inch thick and somewhat larger than the wound is placed over the gauze and cut off squarely at the internatal cleft about an inch above the anus. A two-inch "dryback" adhesive plaster strip is applied tightly across the inferior border of the sponge and carried around to below the anterior superior spines of the ilia. A similar strip is applied at the upper border. The wound is dressed about every fourth day.

CONCLUSIONS

(1) A ten year survey of 288 cases of pilonidal cysts and sinuses from the Surgical Services of Bellevue Hospital, New York City, has been reported, including the follow up of 83 cases.

(2) These congenital malformations originate from faulty skin development.

(3) Trauma is not ordinarily a factor in the onset of symptoms.

(4) The predominant symptoms are due to infection.

(5) The pathologic picture is that of acute and chronic inflammation of skin.

(6) Spinal, caudal, and local anesthesias are definitely contraindicated.

(7) Delineating reagents in the operation have not reduced the incidence of recurrence.

(8) Complete excision and packing is the method of choice.

(9) A method of dressing is suggested.

NOTE.—We wish to express our appreciation to Dr. Arthur M. Wright, Dr. Guilford S. Dudley and Dr. John A. McCreery for their courtesy in allowing us to use the cases from their respective services at Bellevue Hospital; and to Dr. Douglas Symmers, of the Department of Pathology, for permission to examine the microscopic sections from these cases.

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BRIEF COMMUNICATIONS AND CASE REPORTS

AMPUTATION FOLLOWING "PAVAEX" TREATMENT

REPORT OF A CASE

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THAT the method of treating vascular diseases of the extremities by means of the passive vascular exerciser ("Pavaex") as suggested by Reid and Hermann may not be entirely devoid of danger is exemplified by the following case:

CASE REPORT

Mrs. A. A., aged 61, had a midthigh amputation of the right leg at the Johns Hopkins Hospital in 1933 because of gangrene. The diagnosis was arteriosclerosis. Patient had suffered from hypertension for many years.

In August, 1934, her left foot began to hurt and she was admitted to the West Baltimore General Hospital where the intravenous salt solution treatment was given. When this failed to give relief she was referred to another hospital for the "Pavaex" treatment and it was following this that she began to have pain, redness and swelling on the inner side of the leg between the knee and the ankle. She says she called the attention of the doctor in charge to this condition but he said it was of no consequence and continued the treatment. When the leg became still more painful she gave up the treatment of her own accord.

On October 23, 1934, the patient first came under my care. At that time the left foot was a little cool and slightly edematous but there were no ulcers present. There were a number of enlarged superficial veins over the ankle and inner side of the foot. Running up along the inner side of the leg, however, there was a very definite and extensive area of cellulitis along the course of the internal saphenous vein—which was thrombosed—extending practically from the ankle to the knee. This area was red, inflamed, and definitely sensitive. Patient complained it was so painful she could not sleep at night. There were two small dry black patches of skin necrosis in the center of this inflammatory area about one inch apart, one about the size of the end of one's finger and the other about half as large. No arterial pulsations were to be felt in the leg—not even in the femoral at the groin. Blood pressure 210/110. Diagnosis: obliterating endarteritis.

Patient was admitted to the Hospital for the Women of Maryland where, with rest in bed and continuous warm, wet compresses, the condition began to improve and for a time it looked as if it might resolve itself. After several weeks the process became stationary and then slowly but surely began to get worse. The patient was restless at the hospital and was sent home for a while, following which she was persuaded to go into the West Baltimore General Hospital. The two little areas of skin necrosis began to get larger and became definitely ulcerated. The cellulitis, too, began to spread and the patient ran an irregular temperature. All this time her foot remained free from gangrene or ulcer, although it did become slightly more edematous.

The patient insisted on going home. She was there but a short time when the big toe became dark and later on definitely gangrenous. Furthermore, she was suffering considerable pain and a chronic bronchitis was becoming definitely worse. The subject of amputation had been considered some weeks previously but the patient's family would not consent to it. Finally it became obvious that life could be saved only by amputation. She was sent into the Sinai Hospital, where, under evipan anesthesia, a midthigh amputation was done. No tourniquet was used and outside of the great vessels hardly a ligature had to be applied. Her convalescence was uneventful.

This case is reported because such occurrences as this should be brought to the attention of the profession. I personally feel that the "Pavaex" machine is a distinct advance in the treatment of vascular diseases of the extremities, but it is a new departure and the bad as well as the good must be known and recognized in order to evaluate its proper place in our armamentarium. It may very well be that the "Pavaex" treatment in the above-mentioned case had nothing to do with the result; possibly it was purely a coincidence that this patient developed a cellulitis following its use. The probabilities, however, are that this was not the case, and certainly the operator of the machine should have stopped using it on this patient when his attention was called to the obviously increasing cellulitis.

It would seem that not all vascular diseases are suitable for this treatment and that until its limitations are known one should use great care and discrimination in the selection of cases for its use. It would further seem that any cellulitis or phlebitis of an acute nature is a contra-indication, in the first place, and if it occurs during the treatment it should serve as a warning that the treatment had best be discontinued, at least for the time being.

NONTRAUMATIC HERNIA THROUGH THE RIGHT PARASTERNAL FORAMEN OF MORGAGNI*

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AND
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HERNIATIONS through various parts of the diaphragm have been reported in such increasing numbers in recent years that the addition of a single case report would seem to have no justification. However, the present case offered such difficulty in diagnosis that it warrants record.

CASE REPORT

The patient, a widow of 57, was admitted to the Surgical Division "E" of the Hospital of the University of Pennsylvania July 21, 1933, complaining of indigestion. Seventeen months previously she had been operated upon for acute appendicitis and at that time it was learned that for many years she had had attacks of indigestion, characterized by a feeling of fulness in the epigastrium and gaseous eructations after meals.

* Read before the Philadelphia Academy of Surgery, November 5, 1934.

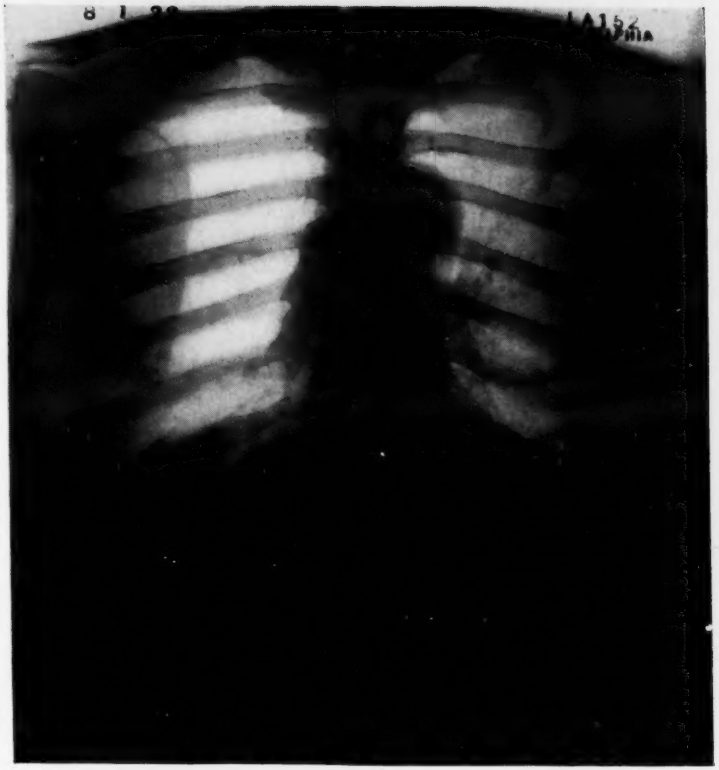

FIG. A.

FIG. B.

FIGS. 1 A and B.—Anteroposterior and lateral radiograms of chest showing large circumscribed lesion in lower part of right chest, located anteriorly just to the right of the midline and in the costophrenic sulcus.

For seven months after her appendectomy she was in good health but then again began to experience increasing epigastric distress. She took soda in large amounts with variable relief, and she was so uncomfortable after eating that she would frequently skip a meal. For three months before her admission in July she had frequent attacks of pain in the right upper quadrant of her abdomen which would radiate to her back and the top of her shoulder. This pain was nagging in character and there was tenderness of the painful area. The pain was more intense at night. Tomatoes and starchy foods always caused indigestion.

Examination on admission showed an obese white woman who was in no apparent distress and did not look sick. There was tenderness under the right twelfth rib and in the left hypochrondrium. No masses and no organs were felt. Except for varices of both legs there were no other abnormal findings. Laboratory studies were all negative for any disease of the biliary or gastro-intestinal tracts. Van den Bergh reaction of the serum showed a negative direct reaction and the indirect showed 0.2 units of bilirubin; icterus index was 5; duodenal drainage gave a good B fraction and the microscopic examination was negative. The cholecystogram was interpreted as showing no disturbance of gallbladder function. Blood Wassermann was negative. Hemoglobin, 87 per cent; white blood cells, 7,400; and blood urea nitrogen 22 milligrams per 100 cubic centimeters. Urine showed occasional leukocytes on microscopic examination. The gastro-intestinal radiographic studies revealed no evidence of disease of this tract but there was observed a large lesion in the right lower chest. Radiograms (Figs. 1A and B) were then taken and reported as follows: "There is a circumscribed lesion located anteriorly just to the right of the midline and in the costophrenic sulcus which does not appear to be inflammatory, does not restrict the movement of the chest walls or the domes of the diaphragm. It may be due to a primary tumor or an echinococcus cyst." At the 24-hour examination it was noted that there was no connection between the colon and the mass of the right half of the diaphragm. At her request she was discharged from the hospital to return at a later date for operation.

The patient was readmitted September 19, 1933. She had not had as severe pain in the upper abdomen but had experienced pain in the right lower back and had had severe headaches. Examination at this time showed her blood pressure to be 90/80, hearts sounds faint. Deep pressure in the epigastrium and under the right costal margin elicited pain in the right back just to the right of the twelfth thoracic vertebra. Bronchoscopy showed deformity of the right stem and lower lobe bronchus which was thought to be due to extrabronchial causes. With the thought that the lesion might be due to an echinococcus cyst a complement fixation test for this disease was done with negative results. Blood studies were again normal. Following the induction of an artificial pneumothorax for diagnostic purposes roentgenograms of the chest with the patient erect and with her lying on her left side were made (Fig. 2). Two distinct shadows were seen at the right base. One presented a stringy appearance and corresponded to the fissure between the right middle and lower lobes. It was thought to be due to pleural thickening the result of an old inflammatory process. The lower and larger shadow appeared to be in the partially collapsed lower lobe. The film taken with the patient lying on her left side showed a definite change in shape of the lesion of the right lower lobe and that it merged with the heart shadow. There was seen an irregularity in the outline of the dome of the right diaphragm which was thought to have the appearance of an individualization of the costal components of the diaphragm. The lung lesion was thought by the roentgenologist to be an atelectatic and cirrhotic process in the right lower lobe due to a long-standing bronchial occlusion which was probably of inflammatory origin. Because of these conclusions the patient was again allowed to go home.

Once more, however, she had numerous attacks of pain in her abdomen. Now they were sharp in character and radiated around the right costal margin and localized in the superior surface of the right shoulder. Heavy lifting brought on attacks but they

also occurred at night and she would be aroused from sleep. Deep breathing accentuated the pain. On November 11 she again entered the hospital. Examination showed slight tenderness on deep pressure in the right costovertebral angle. Pressure in the left upper and right lower quadrants caused pain in the tender right upper quadrant and radiation of the pain to the right shoulder. Due to the persistence of her symptoms and to the uncertainty of the lesion in her chest an exploratory thoracotomy was decided upon with the diagnosis of pulmonary cyst leading the list of probabilities. In preparation for the operation a partial pneumothorax was again established and the diaphragm was temporarily paralyzed by crushing the phrenic nerve and its accessories in the neck.

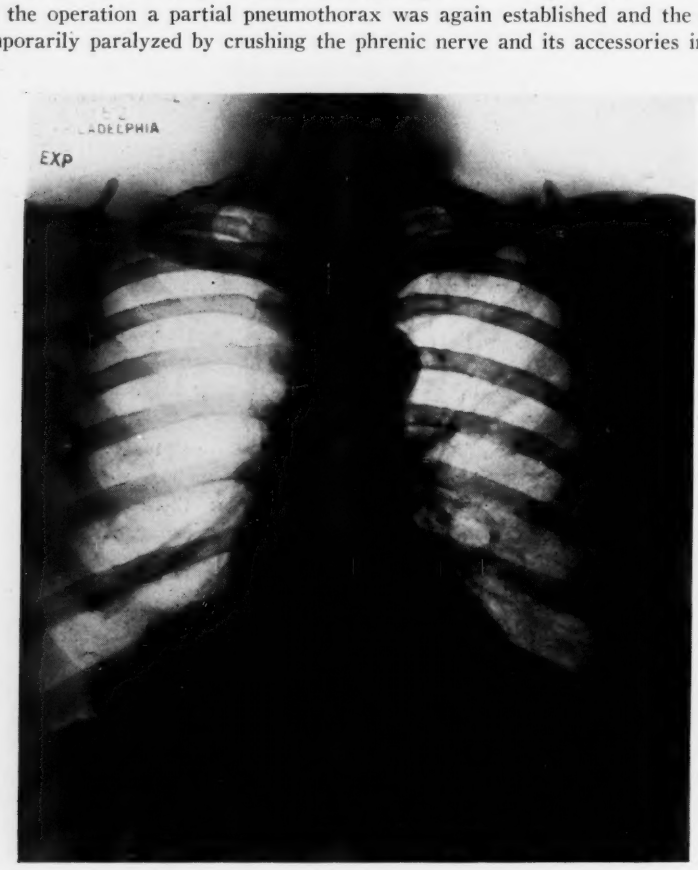

FIG. 2.—Anteroposterior radiogram of chest following partial pneumothorax. There is about 50 per cent collapse of the right lung. There are two distinct shadows seen at the right base. One of these presents a stringy appearance and in position corresponds to the interlobar fissure between the right middle and lower lobe. This shadow is evidently due to pleural thickening of inflammatory origin. The lower and larger shadow appears to be in the partially collapsed lower lobe.

Another roentgenogram (Figs. 3A and B) of the chest was reported by Doctor Pendergrass as follows: "In some respects the lesion has the appearance of a pleural tumor or a pleural cyst. Fluoroscopically the lesion does not move with respiration. We would advise a barium meal before any contemplated operative procedure to rule out the possibility of a right-sided hernia of the dome of the diaphragm." Examination after a barium meal was done with negative results. The surgical staff persisted in its diagnosis of pulmonary cyst.

Operation November 18, 1933, by Doctor Ravdin. Under local anesthesia with novocaine, supplemented with nitrous oxide-oxygen, an incision was made through the

was not reported by the roentgenologist.

FIG. A.

FIG. B.

FIGS. 3 A and B.—Anteroposterior and lateral views of chest following paralysis of right dome of the diaphragm. There is elevation of right dome of the diaphragm and partial pneumothorax persists. Note the rounded shadow in anterior portion of chest, free from lung and apparently continuous with diaphragm. This was not reported by the roentgenologist.

fifth interspace from the posterior axillary line almost to the sternum. The fifth rib was divided at its osteochondral junction. A large fatty mass was seen in the region of the pericardium, from which it at first was thought to arise. After gentle blunt dissection this mass was removed. There was then seen a serous membrane-covered mass projecting upward through an oval defect in the diaphragm. The membrane was incised and omentum was encountered. On traction a portion of the transverse colon appeared. The omentum and colon were then returned to the peritoneal cavity. Upon separating the mass from the edges of the opening it was seen that the orifice measured about eight centimeters in length and three centimeters in width and started one centimeter from the anterior attachment of the diaphragm and extended posteriorly. This was, therefore, a hernia through the right foramen of Morgagni, the potential defect between the sternal and costal components of the diaphragm. After defining the edges, closure of the orifice was accomplished by means of an interrupted suture of doubled No. 1 chromic catgut. The pleural defect was then closed with a single continuous suture of the same material. Passivity of the diaphragm facilitated the repair. Pericostal sutures of doubled chromic catgut were then placed at intervals of two centimeters and the transected rib restored to its proper continuity by means of sutures passed through holes drilled in its ends. Before closing the muscle layers with catgut the lung was fully inflated. The skin and subcutaneous tissues were united with interrupted sutures of silkworm gut. Her condition during the operation was uniformly good and remained so at the close. For a few hours after the operation her blood pressure stayed at a low level but this was corrected by means of ephedrine and a transfusion of 125 cubic centimeters of citrated blood. Following this she made an uneventful convalescence. The wound healed by primary intention. There was a small pleural effusion for a few days. A roentgenogram of her chest on December 4 showed a disappearance of the original lesion, an elevated, paralyzed right half of the diaphragm and a very small pneumothorax. She was discharged in good condition on December 9, 1933.

Since then she had had no recurrence of her original symptoms and can now eat anything she desires without having indigestion. She had continued since operation to have twinges of pain which start in the region of her operative scar and radiate upward to her right shoulder. Radiographic examination on November 5, 1934, after a barium meal revealed return of function in the diaphragm and no evidence of her original lesion. The right half of the diaphragm is elevated and its range of motion is not as great as on the left but it is smooth in outline and moves normally.

This then is a case of nontraumatic hernia through the right parasternal foramen of Morgagni in which the symptoms and physical signs strongly suggested gallbladder disease. As the hernial sac contained only omentum no absolute evidence of the true condition could be obtained by roentgenologic studies. Although the correct diagnosis was suggested by one of the roentgenologists an exploratory thoracotomy was necessary to establish it and correct the condition found.

Herniations through the foramina of Morgagni are relatively rare. Hedblom,[1] in 1931, was able to find only 60 cases reported in the literature. He pointed out that as it was at times difficult to tell before operation whether the hernia originated on the right or left side and as these areas are accessible through an abdominal approach that this route should always be chosen.

Harrington,[2] Hedblom[1] and others[3] have drawn attention to the frequency with which the symptoms of diaphragmatic hernia have been ascribed to disease of the gallbladder. In Harrington's 60 cases the preliminary diagnosis was cholecystitis in 29. Somewhat similar figures have been reported by other

observers. Next in frequency is the diagnosis of gastric disease and when one remembers the great preponderance of herniae through the esophageal hiatus and the high percentage of involvement of the stomach in the sac, such a mistake is readily understood. Irritation of the phrenic nerve endings must be a constant factor in these cases and undoubtedly accounts for many of the symptoms ascribed to disease of the gallbladder.

In our case the location and radiation of the pain clearly resulted from stimulation of the right phrenic nerve and probably of the intercostal nerves supplying sensory fibers to the periphery of the diaphragm.

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RUPTURE OF THE SIGMOID BY HYDROSTATIC PRESSURE*

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CASE REPORT.—A male, aged 60, was admitted to the service of Dr. Charles F. Mitchell, at the Pennsylvania Hospital, September 11, 1934. The patient had been constipated for a number of years and occasionally found it necessary to use enemata to promote evacuation. On the day of admission he had gone to the backyard to a water hydrant, of the type found in the backyards of Philadelphia in the poorer districts and to which was attached some three feet of rubber hose. A brass nozzle, such as is used in sprinkling a lawn, was attached to the free end. This was inserted into the rectum and the water turned on. He said that he felt the water running in and after a few seconds had a sharp agonizing pain in the lower abdomen. The nozzle was withdrawn, followed by the expulsion of a small amount of water and feces. He then made his way into the house and collapsed.

On admission the patient complained bitterly of pain in the abdomen which was more marked in the left lower quadrant. The rigidity was boardlike and generalized. There was no obliteration of liver dulness and there was no shifting dulness. There was no audible peristalsis. Temperature was 96.4°, pulse 80, respirations 30, white blood count 10,000, blood pressure 140/80. Rectal examination was negative. A diagnosis of rupture of the sigmoid was made. At operation five hours after the catastrophe the abdominal cavity was found to contain considerable fluid and particles of feces. About 500 cubic centimeters of fluid were aspirated. At the apex of the sigmoid flexure a tear about three centimeters long was found through the anterior longitudinal band. This was closed transversely with a double line of sutures. Two small tears in the serosa and muscularis with herniation of the mucosa were found proximal to this. These were also sutured. A rubber covered gauze drain was placed in the pelvis, and the abdomen closed around it. A tube was passed through the rectum so that its upper end was beyond the suture line. No evidence of disease of the remainder of the sigmoid was found. The postoperative course was uneventful. The rectal tube came out on the fourth day. There was drainage of colon bacillus pus from the wound for two weeks. The patient was discharged on the twenty-second day as well.

* Read before the Philadelphia Academy of Surgery, December 3, 1934.

This is the only case of its kind that the speaker has been able to find but probably this complication is much more frequent than is apparent. It seems unusual that with the frequent use of enemata and colonic irrigations, rupture of this organ appears to be so infrequent. The pressure of an ordinary enema is insufficient, but the pressure of colonic irrigations might conceivably produce rupture of the sigmoid.

It would seem that when the rectum is straightened out because of distention with fluid that the lower sigmoid would become kinked. This would cause a complete blockage at this point. Further distention would probably produce a rupture at the apex of the sigmoid. The same sequence of events would not take place if a gas such as air were used.

In 1911 E. Willys Andrews[1] published a report together with data concerning 11 cases of perforation of the sigmoid colon by air pressure. Burt[2] states that the rupture occurs generally along the longitudinal bands. In a series of experiments completed by him on the human intestine procured at autopsy he found that the sigmoid of a child withstood the greatest amount of pressure per square inch before rupture. This amounted to 11 pounds. Behrend and Hermann[3] reported a case of what was thought to have been a perforation of the sigmoid as the result of a colonic irrigation given by a naturopath. They assumed that it was a perforation mainly because of the incised character of the wound in the wall of the intestine.

REFERENCES

[1] Andrews, E. Willys: Surg., Gynec., and Obst., vol. 12, p. 63.
[2] Burt: Arch. of Surg., June, 1931.
[3] Behrend and Hermann: A.M.A. Jour., vol. 101, p. 1226.

DISCUSSION.—DR. WALTER E. LEE directed attention to the possibility of rupture of the rectum in proctoscopic examinations. Some years ago he had this occur during an examination, and knew of one other case which occurred several weeks before his own. He was using the old type of proctoscope with a sharp edge and the opening a long bevel. After removing the obturator and attempting to inflate with the hand bulb, with his left hand on the lower part of the abdominal wall, he felt the sensation of the air entering the peritoneal cavity. Pneumoperitoneum was demonstrated by roentgenography and within an hour the abdomen was opened and a small wound found in the anterior wall of the rectum about the junction of the rectum and sigmoid. This was closed by sutures, the abdomen closed without drainage, and the patient made an uneventful recovery. The other case to which he referred was not diagnosed until 72 hours after the accident and when the abdomen was opened there was a general peritonitis which proved fatal. At that time he made a search through the literature and could find no references to similar accidents, but they must have occurred, and the possibility should always be borne in mind.

DR. HARRY E. KNOX recalled the case of a man who was lying down outside a mill where he was employed near a compressed air hose and some of his fellow workers inserted a rubber hose in the rectum and turned on the compressed air. The patient came to postmortem several days later, at which time there were multiple lacerations in the rectum and sigmoid.

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GUNSHOT WOUND OF THE HEAD AND OSTEOMYELITIS OF THE SKULL*

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CASE REPORTS

D. T., white male, aged 15, admitted to the Abington Hospital, April 2, 1933. While out shooting the previous week with an air rifle, he suddenly felt a sharp stabbing pain in his left temporoparietal region, and a drop of blood exuded. He suffered no inconvenience, failing to mention his injury to anyone. Forty-eight hours later a lump appeared, and a radiogram revealed a bullet just inside the bone.

Examination showed a small laceration in the left parietal region where the bullet had entered. This was scabbed over and apparently clean. Neurologic examination was essentially negative. Because of the absence of neurologic symptoms and because of the possibility that the laceration might be infected, it was decided to postpone removal of the bullet.

About 72 hours after his admission to the hospital he commenced to develop sensory aphasia and subsequently had a convulsion which commenced in his right hand and involved the entire right side. The aphasia became complete and a right hemiparesis, more marked in the hand and arm than in the leg, was noted. Inasmuch as he was developing signs of intracranial pressure, immediate operation seemed indicated.

A bone flap was made around the point at which the radiogram showed the bullet to lie. The bullet was found lying loose between the bone and the dura. The hole it had made in the bone was clean-cut and no bony spicules had been thrown off. The bullet fell out when the bone flap was reflected. It had expended its force in passing through the skin and the skull bones so that the dura had not been pierced. Four or five large hairs lay on the dura which was very tight and tense but not discolored.

Since the injury had occurred four or five days before, and since in spite of the presence of the hairs no infection had developed, opening of the dura seemed advisable, for no extradural pathology had been discovered to account for his symptoms. Wide reflection of this membrane revealed a tight brain. At about the center of the opening, there was a circumscribed area of contusion on the surface of the brain. On opening the pia at this point and nicking the cortex, an old, leathery mass of clot about six by three by three centimeters in size extruded. This left a cavity extending down into the brain to a depth of about six centimeters. Ragged brain tissue formed the walls of this cavity. With the suction apparatus all the damaged brain was removed and the opening was carefully extended into the ventricle. This was done so that the cavity formed by the clot might be filled by cerebrospinal fluid and thus prevent the formation of a serious scar. No bleeding accompanied this débridement of the brain. The dura was then tightly closed and the bone flap replaced. The skin was sutured in layers with stab wound drainage behind the posterior limb of the flap.

The boy made an uncomplicated recovery. Within 48 hours his speech was much improved and his hemiparesis disappearing. At the end of two weeks, his speech was normal and his right extremities had normal power. The only obvious sequela was complete astereognosis in his right hand and loss of position in the right big toe. Examination 18 months later revealed the boy to be normal in every respect.

OSTEOMYELITIS OF THE SKULL

M. D. W., white, female, aged 12, admitted to Graduate Hospital, January 5, 1934, with the chief complaint of swelling over the right forehead with chronic discharge from the frontal sinus and septic temperature.

Following swimming three months previously she had a frontal sinus infection

* Read before the Philadelphia Academy of Surgery, November 5, 1934.

which finally developed into pansinusitis. She then had a subperiosteal abscess in the midline about five centimeters above the eyebrow which was opened and drained. Subsequently, she developed another subperiosteal abscess about five centimeters behind this and about five centimeters from the midline on the left side. The drainage afforded was not adequate to take care of the situation.

Physical examination revealed a thin, pale child with a small incision just above the hairline in the midline in front from which pus was draining copiously. Neurologic examination was entirely negative. The eyegrounds were negative. Radiograms showed the presence of at least two sequestra with probable involvement of the left frontal bone in the osteomyelitic process. Operation seemed indicated to afford better drainage.

January 9, 1934, under local anesthesia, an incision was made in the midline of the forehead and carried upward about ten centimeters and then laterally to the left to include the left frontal bone and the infected area shown by the radiogram. There were two areas in which infection was most obvious; namely, directly in the midline about five centimeters above the left eyebrow and at about the frontotemporal suture about eight centimeters to the left of the midline. The skin flap was turned back and the periosteum stripped from the bone which seemed to be infected throughout. The sequestra was removed. Before we finished we had removed most of the left frontal bone piecemeal.

There were several extradural abscesses, particularly one that ran backward behind the sequestrum that lay on the left side at the frontotemporal suture. In pursuing the osteomyelitic process downward and forward, we were compelled to remove the roof of the left frontal sinus and establish drainage from this area into the nose. The condition was much more serious and widespread than the radiogram had indicated.

After the bone was removed, no attempt was made to remove the granulation tissue from the dura. The brain pulsated and there did not seem to be any evidence of an intradural collection. The wound was packed with gauze and the skin flap pulled back and secured in two places with silkworm gut sutures. Dakin irrigation was commenced.

Following this procedure, the child's condition improved somewhat. The temperature dropped and became more regular. The wound appeared to be clearing up well under the vigorous dakinization. However, on February 10, there was a change in her general condition. The discharge from the wound became very profuse and a soft swelling appeared to the right of the midline just in front of the temporoparietal suture. Roentgenologic examination at this time showed that the osteomyelitic condition was extending to involve the right frontal bone. Further operative intervention therefore seemed indicated.

February 24, 1934, under vinesthine anesthesia, the skin was stripped back from the bone in the right frontal region and a considerable area of bone in the right frontotemporal region removed. The two soft swellings were incised and the pus evacuated. Necrotic bone was found beneath each. This was widely removed. Our operative procedure on this occasion amounted to practically the same as it had on the other occasion except this time the bone was removed in the right frontal area rather than the left. Some bone was also taken from the left temporoparietal region. The child has very little left of her frontal bones on either side.

Following this procedure the condition improved. However, it was noted that the granulation tissue in the wound was extremely sluggish and of a peculiar grayish color. A blood count and hemoglobin estimation made March 1, 1934, showed that the child's hemoglobin was 28 per cent and the red cells 2,100,000. She was promptly given transfusions of 200 cubic centimeters of blood three times a week for the next six weeks. The effect of the transfusions on the granulation tissue and general condition of the wound was remarkable. After six transfusions her hemoglobin was 75 per cent and her red cell count 4,200,000. The granulation tissue now looked entirely normal; the discharge from the wound was much less; her temperature much more regular and her general condition and disposition much improved. By the end of March, the

osteomyelitis seemed definitely to be under control except for a small area in the right frontal region and a fairly well localized sequestrum in the left temporal region. The temperature was normal and the discharge from the wound much reduced.

On March 29, 1934, we were able to obtain a fairly satisfactory closure of the wound. The tips of these skin flaps were pierced with needles, the left frontal and the right parietal flap and the right frontal and the left parietal being pulled together by gentle traction from rubber bands over the needles. At this time also, two dozen small pinch grafts were placed at various points over the denuded area.

From this time on the wound healed slowly but satisfactorily. The child was discharged May 25, 1934, with the wound entirely dry. The cosmetic result was quite satisfactory, since we had carefully avoided any incision in the forehead below the hair-line except exactly in the midline. This area we had been able to improve by a plastic procedure at our third operation so that that scar was practically invisible.

The area from which the bone had been removed seemed to include the left frontal bone from a point about three centimeters in front of the vertex down to and including the top but not the anterior wall of the left frontal sinus and laterally into the left temporal fossa to a point about one and one-half centimeters above the zygoma. On the right side the right frontal bone was removed down to but not into the right frontal sinus and laterally only to a point about four centimeters above the zygoma and posteriorly up to the frontoparietal suture.

We preserved the child's hair, and at the present time she has a wig which fits over a flat aluminum plate giving protection against any possible unexpected injury to her head.

AN INCISION FOR THE EXPOSURE OF THE VENTRAL SURFACE OF THE DISTAL END OF THE RADIUS AND ITS RELATED STRUCTURES*

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THE incision here presented gives an excellent anatomic approach to the ventral (anterior) surface of the distal end of the radius and its related structures. It was first used for the open reduction of fractures of lower end of the radius with anterior or ventral displacement of the distal fragment or fragments.

Incision.—(Fig. 1) The pisiform bone is located and the surface topography of the flexor carpi ulnaris carefully outlined. The pisiform bone is easily palpated and is on a plane anterior to the other carpal bones. Having noted the above landmarks—the incision begins one centimeter radial or lateral to the pisiform bone and is carried proximally for a distance of five to six centimeters parallel to the flexor carpi ulnaris. Continuing the incision through the skin and subcutaneous tissues, the ulnar nerve and artery are exposed and together with the flex or carpi ulnaris are retracted medially or to the ulnar side. The entire group of flexor tendons are now retracted laterally or to the radial side. Careful snips with scissors or knife of the volar (anterior) radiocarpal ligament readily permits wide separation in the anatomic plane between flexor carpi ulnaris and the other flexor tendons.

* Read before the Philadelphia Academy of Surgery, December 3, 1934.

This approach injures no important nerves or vessels, and avoids opening the sheaths of any of the flexor tendons. Severing the medial segment of the volar radiocarpal ligament permits wide separation between the flexor carpi ulnaris and the other flexor tendons.

The medial segment of the volar (anterior) radiocarpal ligament is the weakest part of this ligament, and its incision in no way interferes with the integrity of the lateral (radial) segment which bears direct relation to the group of the flexor tendons, which are retracted laterally. The entire group of flexor tendons, with the exception of flexor carpi ulnaris, normally lie

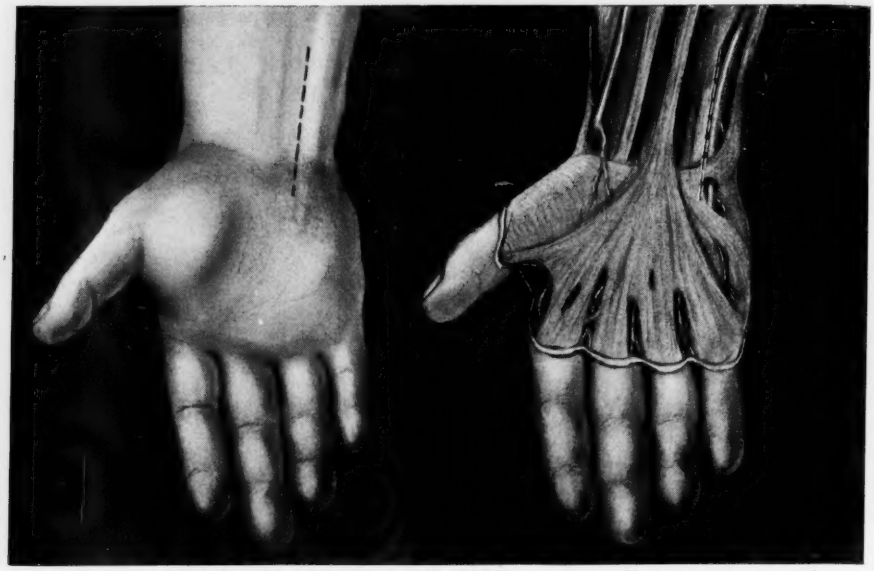

FIG. 1.—Showing site of incision and relation of anatomic structures exposed.

in the smooth depression or hollow formed by the ventral surface of the carpal bones.

Wiedhopf[1] describes an identical incision for the excision of the lunate bone. This approach to the lunate bone seems more direct and less traumatizing than the dorsal approach or the other types of volar incisions that have been used. The difficulty of excision of the lunate through the dorsal route has been noted by a number of surgeons. Orientation from the dorsal aspect is not easy. Cases have been reported in which the triquetrum (cuneiform) or navicular with or without the lunate have been removed in mistake for the lunate. Tillman has advised the insertion of a needle in the lunate bone to aid in its recognition, previous to removal.

The lunate lies obliquely in an upward and inward direction, and by placing the wrist in maximum radial abduction and dorsiflexion with the fingers flexed, the lunate bone is made more prominent and accessible. The anterior surface of the lunate is larger than the dorsal surface, and has only weak ligamentous bands; the dorsal surface is rough and ligamentous. The volar incision allows complete orientation before the radiocarpal joint is opened. And the smooth ventral surface of the distal end of the radius per-

mits easy lateral or radial retraction of the flexor tendons. Once the radiocarpal articulation is opened, the lunate is readily removed.

This incision permits an excellent exposure of the inferior radio-ulnar articulation. The importance of the functional integrity of the inferior radio-ulnar articulation in certain types of ankylosis of the wrist, either of infectious or traumatic origin, is often overlooked. When the elbow is extended, rotation of the forearm is possible by means of rotation at the shoulder joint. With the elbow flexed, rotation (supination and pronation) of the forearm takes place about an axis formed by the ulna and the fifth metacarpal. This rotary movement of the radius about the ulna takes place in the superior and inferior radio-ulnar articulations.

The radius and the hand rotate about this axis as a unit. And at the inferior radio-ulnar articulation the radius and the hand swing about the head of the ulna, much like a door on a hinge. It is a matter of common knowledge that if ankylosis of the wrist is associated with (1) good position of the carpal bones; (2) no loss of pronation and supination; (3) free flexion and extension of the fingers, there is little disturbance in function and such an individual has a good useful hand.

Ankylosis of the radiocarpal articulation showing no involvement of the inferior radio-ulnar articulation does not result in a loss of pronation and supination, since the radius and carpal bones rotate as a single unit. On the other hand, if involvement of the inferior radio-ulnar articulation develops in addition to the ankylosis of the wrist, the resultant loss of pronation and supination produces considerable crippling in the function of the hand.

The type of ankylosis of the wrist that is associated with a loss of supination and pronation, for which no cause other than involvement of the inferior radio-ulnar articulation is found, pronation and supination may be reestablished, either by an arthroplasty of this articulation or artificial pseudarthrosis proximal to the head of the ulna, or by a subperiosteal resection of the ulna (head and neck) leaving the styloid process intact (Darrach's operation).

REFERENCE

[1] Wiedhopf, O.: Deut. Ztschr. f. Chir., vol. 235, p. 384, 1932.

DISCUSSION.—DR. B. FRANKLIN BUZBY said that personally, he had had no experience with this incision. There were three or four points which Doctor Lipshutz had brought out with which he was not in accord. First, concerning the excision of the semilunar bone. There are three indications for this: the first is dislocation, the second Kienboch's disease and the third fractures. In Kienboch's disease when dealing with the soft bone we should make a dorsal incision as is commonly done between the extensors of the fingers, outline the semilunar bone and cut its stronger ligamentus attachments on the dorsum from the weaker ligament on the flexor surface and deliver the bone intact without fragmentation, thereby doing less damage to the wrist joint as a whole. When dealing with dislocations of the semilunar the median nerve is frequently damaged. Through the incision as described by Doctor Lipshutz it cannot be exposed. In that case the flexor

midline incision would be the better. The mistake to which he refers, namely that the cuneiform and the semilunar are sometimes removed in place of each other, it would seem that if the incision is properly placed dorsally this mistake is not so apt to happen. I could not quite understand his description as to the use of this incision in removal of a segment of the lower ulna to permit pronation and supination of the forearm in low radio-ulnar synostosis. It seems to me that if we want to take a section out of the lower ulna it is going quite a bit afield to approach it through Dr. Lipshutz's incision when it can be approached safely by an incision directly over the bony operative site itself with easy retraction of contiguous structures. For fractures of the lower end of the radius with anterior displacement it seems the ideal procedure. For other fractures of the lower end of the radius it would seem too far to the ulnar side.

ANTERIOR PERINEPHRITIC ABSCESS*

ABDOMINAL AND THORACIC COMPLICATIONS

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CASE REPORT.—A man, 21 years old, was admitted to Roosevelt Hospital complaining of pain in the right lower abdomen of seven days' duration. He had been awakened at night on the date of onset with a sharp, stabbing pain in the right lower abdominal quadrant which radiated slightly to the back in the right lumbar region. No nausea or vomiting. The pain lasted a few minutes and disappeared, only to recur two days later. His only complaint on admission was of soreness in the right lower abdomen and the right flank which had persisted since the onset of his illness.

Examination showed no abnormalities except some muscular spasm in the right side of the abdomen. There was some tenderness at McBurney's point and also at the costal margin and extending into the right epigastrium. Slight costovertebral tenderness was noted on the right side.

On admission September 29, 1933, his temperature was 104.2° which, on the day following, subsided rather rapidly to normal. Blood count, 20,000; polymorphonuclears, 91 per cent. Urine showed an occasional white blood cell, rare red cell and repeated examinations the first few days following his admission showed no change in this respect until the third day when the number of white cells was somewhat increased. Notwithstanding his normal temperature, his blood count during the first few days continued high.

He continued to complain of the pain in the right upper quadrant which on the fourth day became much more severe and constant. He vomited several times a thin, yellowish material. There was, at this time, some diminution of breath sounds on the right side of the chest but no râles or friction rubs. The upper rectus was spastic and the maximum tenderness was discovered in the gallbladder area. It was thought that he was suffering from gallbladder colic although right pleurisy or possibly a kidney condition could not be excluded.

The gallbladder could not be visualized on roentgenographic examination. Cystoscopic examination showed nothing abnormal and a pyelogram was normal.

Operation through a right rectus incision on October 7. No abnormality was found in the stomach, gallbladder, duodenum, appendix or liver. An induration was felt near the posterior inferior margin of the liver and here the finger suddenly broke into a large abscess cavity lying between the kidney and liver extending up some distance beneath the ribs. A stab wound was made opposite the end of the eleventh rib in the flank and a tube placed directly into the abscess cavity. Another tube was brought out from the subhepatic area in the anterior abdominal wall which was closed above it.

* Presented before the New York Surgical Society, January 23, 1935.

The patient's temperature rose shortly after operation to 105°, gradually diminishing during the next ten days to 102½° at night and 100° in the morning. During this time the patient appeared quite septic and received numerous infusions and transfusions. The drainage from the anterior wound rather rapidly diminished and the tube was withdrawn. From the posterior wound the drainage was massive, containing large flocculi as well as pus and it was felt that some necrosis of the adjacent liver tissue was occurring. Blood culture was sterile. The signs in the right chest continued to suggest right lower lobe infiltration and the presence of some fluid. Roentgen examination of the chest suggested pneumonic lesion of the right lung with pleural fluid. A thoracentesis on his twelfth postoperative day showed no pus. On the sixteenth postoperative day a roentgenogram showed increased amount of fluid in the pleural cavity with displacement of the heart to the left side. The patient's temperatrue had begun to rise again reaching 105°. Another thoracentesis was done and about ten cubic centimetres of foul smelling, creamy pus was obtained. A later puncture obtained only clear fluid. The original stab wound was enlarged and the finger introduced into the abscess cavity. This contained a large quantity of thick pus, and it was felt that the exploratory puncture of the day before had probably entered this cavity rather than the pleural space.

The temperature ranged to 103° for five days then rose to 105° during the next two days. Thoracentesis in the fifth interspace posteriorly obtained 900 cubic centimetres of thin, slightly cloudy fluid. Culture from this was negative. The patient seemed much improved by this measure and continued for a week with temperature running only to 102°. Roentgen ray now showed some elevation of the right leaf of the diaphragm, and it was felt that there might have been some extension from the abscess into the subdiaphragmatic area. As the patient's condition continued to improve no further intervention was undertaken. Lipiodol injection of the sinus tract leading to the abscess showed no communication above the diaphragm.

An exploratory puncture of the chest was again done December 4, in the seventh interspace in the anterior axillary line and about 40 cubic centimetres of thick, creamy pus was obtained. On December 8 an attempt was made to introduce a needle again into the pocket previously discovered, without success. The original drainage wound in the side was enlarged again and the finger introduced. No pus was encountered. Exploration beneath the diaphragm well up above the dome of the liver showed no pus in this area. A drain was introduced. Owing to the patient's condition, it was decided not to do a complete thoracotomy at this time but a segment of the rib was removed and the wound packed open.

The patient's condition continued to improve without further manipulation, the temperature reaching normal December 25: He was discharged January 18, 1934, 102 days after his first operation. His subsequent course has been entirely uneventful.

This case is shown to illustrate: (1) A difficulty in diagnosis which may arise in distinguishing between an anterior perinephritic abscess and certain abdominal conditions—notably retrocolic appendicitis, acute cholecystitis, and liver abscess. (2) Complications, abdominal and thoracic, that may result from such a condition.

DISCUSSION.—DR. A. HYMAN felt that the diagnosis of anterior perinephric abscess was frequently overlooked, as illustrated in the case just presented which was mistaken for a gallbladder condition. When the abscess is on the anterior surface of the kidney, the renal symptoms may be negligible and the upper abdominal symptoms so pronounced as to closely simulate gallbladder pathology. The urologist sees these cases more frequently, and with refined methods of diagnosis is more apt to make a correct diagnosis.

He wished to know if the radiogram showed obliteration of the psoas muscle outline, or deviation of the spine. These two radiographic changes

are of considerable diagnostic import. Pyelography, either retrograde or intravenous, very often gives additional diagnostic data. The pyelogram may show only very slight changes, evidenced by compression or distortion, and unless properly interpreted may be overlooked.

The urinary and cystoscopic findings may be entirely normal, also kidney functional tests. An additional aid in diagnosis is the culture of the urine. Practically all of these cases are *Staphylococcus aureus* infections and a positive culture from the suspected kidney with negative findings from the other side is of diagnostic value.

He inquired whether the pulmonary complication was due to a perforation of the abscess through the diaphragm. Two years ago, Doctor Kingenstein showed a man of 62, who was operated upon for a left perinephric abscess secondary to a calculous pyonephrosis. Thirty-two days following operation, during the course of a routine irrigation of the lumbar sinus with acriflavine, the patient experienced a paroxysm of coughing and expectorated a large amount of yellow stained watery fluid. It was evident that the patient had a communication between the perinephric region and the bronchial system. This was confirmed by lipiodol injections. Following a secondary nephrectomy, the patient slowly recovered from his infection and the bronchial fistula apparently closed. This type of pulmonary complication due to a perforation is extremely rare, only three cases having been reported in the literature in the last thirty years.

DOCTOR CUTLER said that the roentgenograms did not clearly demonstrate psoas obliteration. They had reviewed the films subsequently with that in mind, but the psoas showed no more vaguely on one side than on the other. There was a slight deviation of the spine which was thought might be accounted for by some irregularity in the patient's position on the table, especially in view of an entirely normal pyelogram. There was *Staphylococcus aureus* in the pus obtained from the original abscess. In the urine, there was only a somewhat more than normal number of white cells and only a few red blood cells before operation. There was no pus until three days after operation. As to the extension into the chest, it was thought to be by contiguity rather than by anatomic extension. At one time in his prolonged septic chest complication the radiogram showed what purported to have been a small hydro- or pyopneumothorax. They had injected lipiodol through the drainage wound tract to see if there was extension into the pleural cavity, but no sinus was demonstrable.

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